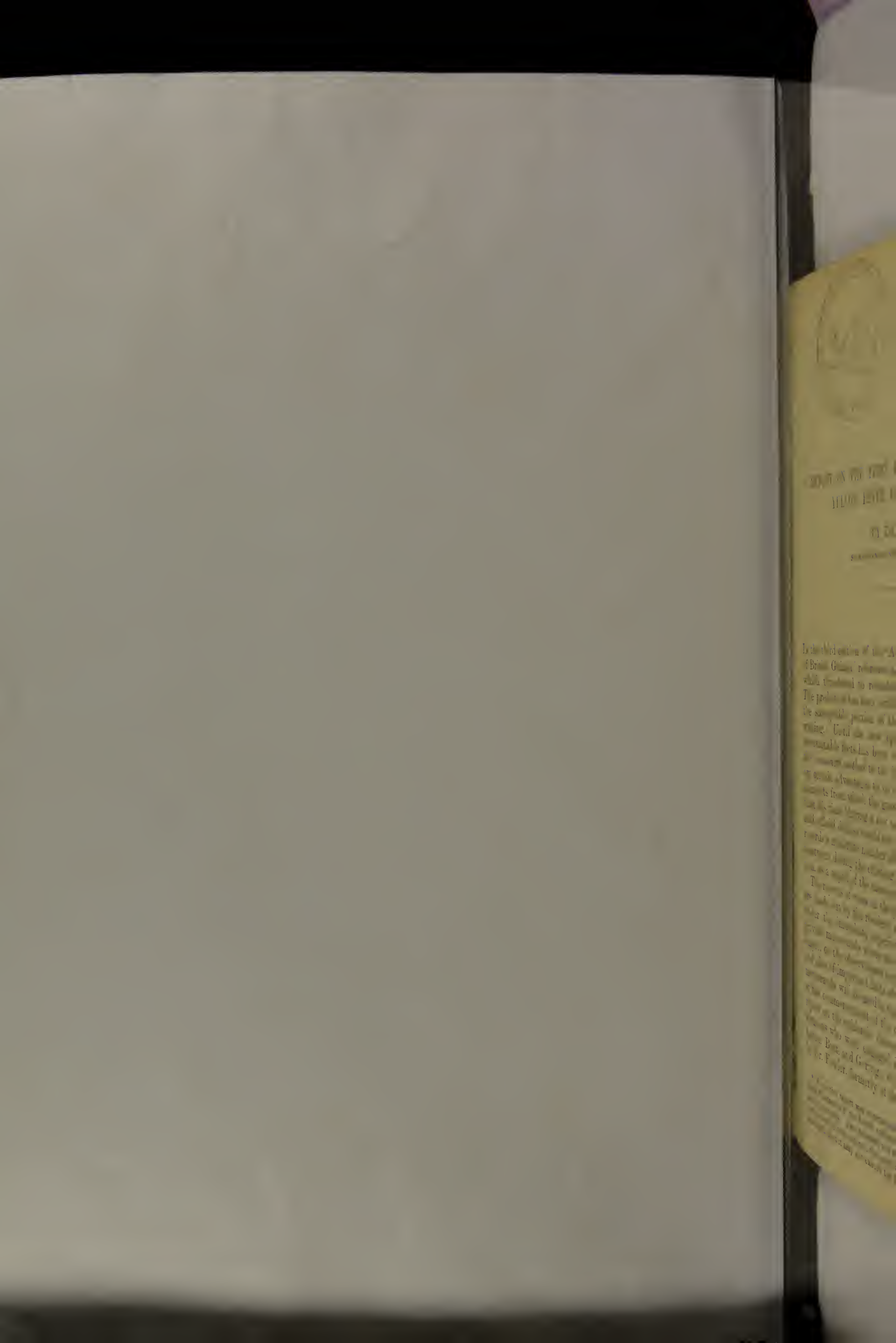

REPORT ON THE FIRST EIGHTEEN MONTHS OF THE FOURTH YELLOW FEVER EPIDEMIC OF BRITISH GUIANA.

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CHAPTER I.

In the third edition of the 'Account of the last Yellow Fever Epidemic of British Guiana,' reference is made to the outbreak of a new epidemic which threatened to resemble that of 1837 in direction and severity. The prediction has been verified; and the pestilence continues to afflict the susceptible portion of the community till the time of the present writing. Until the new epidemic has passed away, and the cycle of ascertainable facts has been completed, it would be premature to apply the *numerical method* to the investigation of its phenomena; but as there are certain advantages to be derived from a detail of individual cases, the elements from which the grand averages are obtained, the writer has felt that his time (during a few months' leave of absence from his professional and official duties) would not be misapplied in selecting from the hospital records a moderate number of cases of those who had been admitted for treatment during the existing epidemic, and arranging them for publication, as a sequel of the monograph above referred to.

The reports of cases in the public hospital of Demerara and Essequibo are made out by the resident surgeons at the bedside of the patients, and under the immediate superintendence of the writer. He also keeps private memoranda where the hospital reports seem to him not sufficiently ample, or the observations recorded admit of doubt as to their accuracy; and also of important facts observed by him in private practice. These memoranda will be used in the form of preparatory notes, where applicable, at the commencement of the cases, and in the construction of a general report on the epidemic disease now under consideration. The resident surgeons who were engaged in reporting the cases were, Drs. Driessan, Levin, Butt, and Goring; and a few very interesting cases are reported by Dr. Fowler, formerly of this hospital.

* When this report was commenced, it was intended as an introduction to a volume of cases illustrative of the fourth epidemic of the colony, and selected from the records of the public hospitals. Two hundred and sixty-nine cases were copied out; but the plan of publication having been altered, the cases are omitted for the present, and the report somewhat abridged, that it may not exceed the limits suitable for the pages of a periodical.

The *microscopes*, used were, one of Pritchard's for ordinary work, and one of Ross's for minute observation. The *test paper* was Griffin's neutral tint, of the *old stock*. Each patient was furnished with three vessels, and the ejections were kept scrupulously apart till after examination. The double test for albumen was always applied, and sources of fallacy sedulously attended to.

The arrangement of the cases will be into *aborted*, *recovered*, and *fatal*, and cases occurring among the *dark races:* each division including incidentally relapse cases, and cases of second or third attack. The number of cases given in each division will bear no relation to the actual number of such cases which are to be found in the hospital-books—that would be a quantitative analysis, which must be reserved till the close of the epidemic. All the complete fatal cases will be given—that is, all those in which there has been a post-mortem examination. Almost all the cases in which recovery has followed black vomit, will also be given. Cases of special interest, no matter in which division, also will appear. The *aborted* cases, being short, might admit, with convenience, of a lengthened list; but there is so much uniformity in the march of such cases, that a long array of them would be quite unnecessary for the instruction of the student. Sometimes, however, in this class, the implied definition of them will be found to shift. Thus, cases will occasionally be found that are at once arrested by the larger dose of calomel and quinine, and a purgative; sometimes, auxiliary treatment, and slight *after*-treatment, are required in addition. Very seldom do the symptoms in this class run as far as *albuminous urine*, and never to the stage of *acid elimination*, which would exclude any such case from the category of aborted cases.

Although the hospital cases are appealed to as illustrations of the statements and opinions of the Report, it is to be recollected that the hospital reports were not made by me, but by independent observers; and that though, therefore, they have their peculiar value as such, that perfect appositeness is not to be expected, nor the development of salient points, nor the sufficiency and effectiveness of individual cases, as if the doctrine and testimony came from the same source; still I believe that the evidence will be found ample and satisfactory.

The Georgetown Hospital, or, as it is known by ordinance, the Public Hospital of Demerara and Essequibo, besides other special branches, is divided into seamen's and colonial departments, and the wards of each are in separate buildings. The patients of the latter department consist chiefly of the immigrant labouring population, of which a large proportion are Portuguese or Madeirans, who are highly susceptible of yellow fever. Most of the cases are admitted in a late stage of the disease, owing, in part, to the insidious nature of the malady, to its being mistaken for harmless intermittent fever, as well as from apathy and indifference of the patients and their friends. The seamen have a superstitious dread of hospitals, which is, of course, intensified during the ravages of an epidemic. But, by a salutary law of the colony, the responsibility of obtaining speedy medical relief for sick seamen is thrown on the masters of vessels, respectively. Hence, although the law is often disregarded, the cases from the shipping are generally admitted in better time than

those of the colonial department, their histories are better ascertained, their treatment has a better chance of success; and hence, such cases, chiefly, have been chosen as representative cases in the following collection.

CHAPTER II.

Since the beginning of 1845, the health of the seamen of this port was all that could be desired, and that of the colony, generally, was good. In the months of June, July, August, and September, 1850, mumps became epidemic and epizootic, and was very fatal to cattle. From the 21st of July, 1851, till about the 24th of August following, a malignant influenza swept over the country, and was very fatal to the feeble and dissipated of the Coolie and Portuguese immigrant population. The influenza was, however, almost unfelt by the seamen, and till the end of that year, when yellow fever reappeared in the manner described in the last edition of the 'Account,' the harbour of Georgetown might have ranked among the healthiest in the world; and no disease existed in the colony of which the newly-arrived European or North American need have had the slightest apprehension. The year of the advent of the new epidemic, and the following year (1851 and 1852), were remarkable for the extraordinary average yield of the sugar plantations, being nearly double the average crop. The increase was due almost entirely to the favourableness of the season. From 1849, a great change took place in the distribution of rain over the colony. That year was the acme of the rainy years. Before it, and up till 1851, the rain and dry weather appear gathered up in the meteorological charts in large masses. Since then there had been a less quantity and greater dispersion. During 1851, the rain was so equally distributed over all the months, that no great washing or drying of the country took place. The meteorological characteristic of the weather preceding and accompanying the advent of the new epidemic, therefore, was *the absence of any decided dry or any decided rainy season.* It was favourable to vegetation and agreeable to the feelings; and the minimum temperature of six years occurred in the month of January, 1852 (13th), when the thermometer fell as low as 67·7°. The coincidence of the invasion with the most cool and agreeable time of the year, corresponded in this respect with the epidemic that preceded ours along the windward coast of South America; that of Cayenne having commenced about the end of November, 1850, and that of Surinam about the end of January, 1851.

Although our former epidemic had every appearance of local origin only, that from which the colony now suffers would seem to be the result of some general exciting cause acting consecutively along the south-eastern seaboard of America, which, beginning at the Brazils, passed on to French, then Dutch, then British Guiana—thence to the West India Islands, New Orleans, and, finally, Bermuda. Had the winter not interfered, probably Philadelphia and New York would have been reached. Although, if its diffusion was due to the agency of the trade-winds solely, whose course it followed, the latitude of Bermuda should have been its terminus. The hypothesis of a great epidemic wave, rising in the east, and flowing on westerly, only apparently suffers from a minute inquiry into its course; for, although Demerara was invaded at the end of 1851

while Berbice, which is easterly, or to windward, did not suffer seriously till the end of July, 1852; still, in New Amsterdam, the port and capital of the latter country, two fatal cases occurred as early as February, 1852, and one in May following; and it is to be considered that this town (unlike Georgetown) is situated several miles above the mouth of its river, and may have been caught, so to speak, in the eddy of that great epidemic wave, which so peculiarly affects the sea margins.

Although the present epidemic has been apparently more intense and diffusive than its predecessor, and its origin seems referable to a foreign source, still it affects special localities as before; and the tenements which suffered most on a former occasion, have been again those of its severest visitations. The *focus of intensity*, as indicated by the number of cases and the short period of incubation, is, as was before, the mouth of the river and its east bank. From thence it extended, in unequal radii, to the west bank, the islands in the mouth of the Essequibo river, and the Essequibo coast to leeward; and up the east coast of Demerara to windward, more slowly, and for a shorter distance. About the middle of June, it had reached Plantation Plaisance, about ten miles east of Georgetown; and by August, Mahaica, which seems to have been its eastern boundary, only twenty miles from Georgetown. In the intervening district of Mahaicony, which separates the county of Demerara from the county of Berbice, no case occurred having its origin there. On the 9th of April, a case was admitted to the Colonial Hospital, which had its origin in Camounie Creek, about twenty miles *inland* from Georgetown, and in the air-line of the trade-winds. In November of the same year (1852), the epidemic influence, in an extensive but diluted form, and of only a month's duration, extended to the penal settlement at the junction of the Cayenie and Mayaroonie rivers, about sixty miles inland, and still in the direction of the trades. Prior to this date, a single fatal sporadic case occurred at the penal settlement, of which special notice will be taken hereafter.

The march of the epidemic, its dates and lines of diffusion, would indicate the influence of atmospheric currents on its progress. Outside the boundaries of epidemic influence just defined, there was safety. The danger seemed in some measure proportioned to the nearness of approach to the centre of infection; and several striking instances have occurred of parties descending on a visit from the uplands of the interior, and the uninfected regions of the coast, falling victims to the infection of the town. Within its circumscribed range, the epidemic manifested local predilections; and though some places seemed permanently infected, the lines of infection occasionally shifted, as in the former epidemic; and infected and uninfected localities were temporarily in juxtaposition. Thus the *Marion* of the Clyde sailed from the port in August, after having all hands sick, of whom six died. The *Unicorn*, also from the Clyde, arrived and took her berth, and after lying six weeks, also sailed, without a man sickening. Thus, also, while on Plantation La Penitence and Plantation Houston the mortality of the Portuguese was excessive in January and February, 1852, Plantation Ruimveld, which lies between these two estates, and had as many susceptible subjects on it, scarcely experienced the disease.

Lulls and exacerbations in the general violence and intensity of the

epidemic were frequently observed in its course. The first of these lulls occurred in the last half of the month of March, and the first exacerbation in June. By the end of August, another lull, but of short duration. In February and March, 1853, the epidemic power was intense. It moderated again till June, when it was renewed with great virulence. These lulls in the epidemic were as illusive as the lull of symptoms in the fatal progress of the disease; and it was often my painful duty to discourage the hopes that were so eagerly entertained by the authorities and the public, of the entire and speedy disappearance of the epidemic, and to resist, with apparent pertinacity, the repeated proposals for the return of the white troops to the military service of the colony. During the periods of exacerbation, "threatenings" in the hospital became numerous. These were cases which occurred in the persons of patients admitted for other ailments, and who, during these periods, seemed to be in an *explosive* condition. Such cases presented all the appearances of an invasion of the disease. They were almost all aborted by prompt treatment; but there was seldom any record kept of them, unless they resisted one or two doses.

Although the epidemic sprung up at a delightful season of the year, when the general health was excellent, and, perhaps, irrespective of weather, yet in its course it seemed materially influenced by meteorological conditions; and sometimes even diurnal variations were observable in the condition of the whole of the patients in the hospital, which could only be referable to atmospheric causes. A cool, dry, brisk air seemed to have a mitigating effect; while a hot, sultry, close, moist air increased the number of admissions, and aggravated the type of the disease, particularly on its immediately following the other meteorological state.

Although, when the epidemic influence was strong, intermittent fever and its sequelæ disappeared, it sometimes seemed to blend itself with that disease. It impressed itself sometimes on other diseases, and was itself impressible. Although the influenza came suddenly and disappeared suddenly, about the month of August, 1851, it still left some traces of its influence in the manifestations of disease, and might be detected modifying the yellow fever. In Surinam, the two epidemics were contemporaneous; and we find, by the medical report of the chief medical officer of that colony to the governor, that the two formed a completely mixed disease.

A "phlogistic constitution" of the atmosphere was often observed with us, indicated, through the population non-susceptible of yellow fever, by the prevalence of pleuritis, hepatitis, and dysentery. This condition was found to impress itself on the epidemic, and to influence local congestions and determinations in the progress of the disease. During the exacerbation of the epidemic in June, 1853, small-pox became very prevalent, and was suspected in some cases of spontaneous origin. Mixed cases of the two diseases occasionally happened, the small-pox always predominating. Sometimes the yellow fever would engraft itself on the secondary form of small-pox, after the stage of desquamation commenced, and then it had its own sway unmolested. The co-existence of pneumonia and pleuritis with yellow fever, sometimes the one being primary, sometimes the other, was of frequent occurrence, particularly among the

Portuguese immigrants. In the course of the epidemic, several long-standing cases of chronic disease, to the consternation and surprise of the bystanders, terminated suddenly and fatally by black vomit without any preeursory fever. But if the epidemic, as a whole, was subject to modifications and fluctuations, the *early* individual symptoms of the disease were similarly affected. Sometimes the full complement of standard symptoms were present, sometimes they were imperfect or deficient, and sometimes displaced. At one time the diagnostic symptom was the supra-orbital headache. This, in the epidemics of Cayenne and Surinam, seems to have been the constant characteristic, accompanied generally by lumbar pain. At other times, the tongue symptoms alone were diagnostic. Sometimes their equivalent was observed in the fauces and uvula. In the Surinam and Cayenne epidemics our tongue symptoms do not seem to have been at all recognised. These variations and shiftings of the symptoms were not irregular or promiscuous, but *periodical;* and they continued steadily for several weeks together. Towards the end of April, 1852, the tongue symptoms were unpronounced. Towards the end of May, they were well marked. In the middle of September, the early diagnostic was the frontal headache. On the 4th of October, the tongue symptoms were again well marked. On the 15th of December, they were absent, and the head symptoms developed. On the 21st of December, the absent symptoms were unusually numerous. On the 8th of March, 1853, the tongue, eye, and lip symptoms were intensely developed. Intense surface heat, early albumen in urine, and early black vomit, were the character of the later symptoms; and smoky, pale urine, with perfect blood-corpuscles, took the place of the straw-coloured or bilious urine, with its sediment of tube-casts and epithelial matter. Notwithstanding this variation of symptoms, they were never so defective as to prevent the formation of a correct estimate of the nature of the disease with which the practitioner had to deal. The variation of symptoms had sometimes a relation to the mode of accession of the disease. In the diarrhœal or choleroid cases, the tongue and head symptoms were seldom so early or developed. Having thus sketched the circumstances and general habits of this new epidemic, I shall proceed to consider its phenomena as an individual disease.

CHAPTER III.

In general, the testimony of the patient is, that he was quite well up to the date of attack, and he can tell with accuracy when it began. He was probably awoke in the night by a severe pain in his forehead and back, with sickness of stomach and vomiting, and a sensation of heat and thirst. Or he may have had a decided chill with the frontal headache, and a sweaty skin. Or he may have had violent vomiting and purging, with cramps of the gastrocnemii muscles. In almost all cases, however, supra-orbital pain and fever are associated. Occasionally, the headache may have been experienced several days before the invasion; and a fatal case occurred, in which the characteristic headache and a feeling of *mal-aise* and oppression of the præcordia existed thirteen days before the overt invasion. Dr. Levin, the resident surgeon, who died on the third

day of his illness, complained of sore throat and dysphagia (which continued longer than the sore throat), and muscular pains of the neck and chest, for fourteen days before his seizure. These precursory symptoms were at the time ascribed to cold or influenza, which he was supposed to have contracted by exposure to a thorough draught after having been heated by violent exercise. In the fatal case of Mr. R., at Messrs. Gray, Cunningham and Co., *vomiting* alone was the first symptom complained of. It commenced at night, but he got up next day and attended to his duties in the store, and ate a hearty dinner afterwards. The following night the vomiting returned about the same hour, with fever and intense frontal headache.

The supra-orbital headache is a truly valuable diagnostic symptom in old residents, in the dark races, and those of low susceptibility, and in those cases of diseased complications which would tend to suppress the development of capillary irritation. In the case of W. Munro (Seaman's Hospital, November, 1852), it *was the only obvious symptom* of his attack, the correct diagnosis from which was subsequently demonstrated. As has already been said, it is a most valuable premonitory symptom, giving notice often several days before the actual seizure or overt manifestation of the disease. This headache, and the punctuated tongue, were the signs by which epidemic taint was detected in some cases of intermittents, and which would not yield to ordinary treatment of such cases. This headache is unlike the temporal and general headaches of intermittents, but like it in being often associated with lumbar pain. It is generally relieved speedily by the first or second dose of medicine. But it seems normally to belong only to the formative and febrile stages of the disease, and subsides spontaneously in the middle and late stages. Its etiology is obscure, and the causes may be compound. The pain during the fever is sometimes too intense to be referable alone to any imaginable degree of capillary vascularity of the lining membrane of the frontal sinuses. It is probably aggravated by the hydraulic pressure of the blood in the brain during the fever stage. The pain is sometimes described as in the orbits, more rarely in the upper part of the forehead, and occasionally as extending to the occiput. I have seen cases in which it was instantly relieved by the vomiting of bile. An increase of temperature over the forehead generally accompanies this characteristic headache.

The practitioner having noted the symptoms derivable from the testimony of the yellow fever patient, will observe a *specific capillary irritation* showing itself in the flush of the face, as characteristic as the hectic of phthisis or the fuliginous complexion of typhus. This suffusion generally occupies a zone over the eyes, and about an inch above and below them. The eyes are injected, like those of a person *just awake*, but generally without any lachrymation or photophobia, although the injection may be as intense as in ophthalmia. Sometimes the irritation extends to the palpebra, to one or both, and sometimes only one eye is affected, but that so violently, as if the patient had been *stung* or received a blow on the eye before admission, as in the case of Cash (S. H., 17th May, 1852). The nares also may be found injected, with a coarse vascularity. The lips may be crimson or vermilion coloured; the tongue scarlet at tip and edges. If the fauces be examined, the roof of the hard palate will

sometimes be found covered with a coarse network of capillaries, and this reticular vascularity extends to the uvula. If the practitioner has already been satisfied as to the nature of the case, this examination of the fauces should be omitted, on account of the *vomituritis* generally induced by pressing down the tongue preliminary to the observation. The following entries in my memoranda-book are the first notices, I believe, of this sign: "To-day I was called to see a Portuguese who was ill with yellow fever. The frontal headache and vascularity of eyes were sufficiently marked, but the tongue trifflingly so. He was facing the light of the evening sun, when I asked him to show his tongue, and he opened his mouth so wide that I saw down the posterior fauces. The top of the pharyngeal pouch was unusually red and vascular. *Mem.*—This appearance to be looked for in Seaman's Hospital, 6th February, 1852."

"The *throat sign* confirmed by observation at Seaman's Hospital, 9th February, 1852."

"*New sign of yellow fever.*—The examination of the throat to observe vascularity and redness, causes nausea and retching when the tongue is pressed down by the finger or spatula. This unusual sensitiveness of the stomach enhances the value of the sign, 18th February, 1852."

If a careful examination be extended to the chest, a sub-cutaneous rash may sometimes be observed, which occasionally extends to the arms and abdomen. This efflorescence of the skin is the rarest manifestation of capillary irritation in yellow fever.

Such are the *external symptoms* which declare themselves to the eye. They may be all present, or the majority may be absent. Those generally present are the injected eye, and red-edged and tipped tongue. But though I have never required to investigate further for this class of symptoms, on the first or second day of disease, in any case of yellow fever, I could conceive the possibility of an instance of genuine yellow fever presenting none of these appearances; and, in that case, I would examine the scrotum and anus and rectum with a speculum, for a similar sign: and then I would expect no vomiting, and would look to the cæcum in the post-mortem examination for those appearances usually seen in the stomach. For all practical purposes, the tongue-sign is by far the most important, and is the seat of the most interesting daily observations during the whole course of the disease. All these external symptoms may be more or less intense, and their number and degree constitute an important element of prognosis. They are much more distinctly manifested in the European or North American than in the Madeiran, and are at the *minimum* in anæmics from intermittent fever, or where pericarditis is co-existent. The redness of the tongue varies in tint and in arrangement. In some cases it is confined to the tip; in others, the tip and edges are simultaneously affected. I once observed it in the under surface, near the frænum. In other cases, again, the redness on the tip and edges, or on the surface, is in dots or punctuations: sometimes they are few in number and elevated, and occupy the fungiform papillæ. The punctuated redness is a mild indication, and is the form generally in diseases merely tainted with the prevailing epidemic, or in the formative stage, when the frontal headache exists without any overt seizure having yet happened. In such cases, if the disease proceeds, on the second or

third day the punctuations will have *fused*, and a continuous crimson appearance will be formed. On the fourth or fifth day, if the disease has been aborted, or subsided after acid elimination, the dottings will be restored, and then speedily disappear. These little speckles are sometimes faint red, and sometimes fiery; but in all cases are distinctly discernible through any fur that may be on the tongue. At the tip and edges, the usual site of morbid redness, the tongue is always clean. When the dots project above the surface, these sparse papillæ are always crimson or scarlet. The general appearance of the tongue, however, is *uniform* redness of the tip, or tip and edges together; and the general shape is inclined to be compressed, or wedge-shaped. If we might describe by differences—the contrast with a yellow fever tongue would be a broad, flat, flaccid, pale tongue, round at the tip, and broad and indented, or thin and diaphanous at the edges—a tongue, as if slightly œdematous, and circulating little red blood in its capillaries. Such a tongue, in a yellow fever patient, would be "clean gain." The next change which appears in the yellow fever tongue refers to the upper flat surface. The fur, which at first is smooth and uniform, seems as if it had been *curdled*, and lies on the surface in innumerable little greyish-white wavy flakes. This appearance is most observable when the tongue has been broad and sordid. In the case books of the hospital it is called *curdled*—it might be called pilose, or villous. But its first name is derived from its close resemblance to milk acted on by acids, and from the idea, once entertained, that it was perhaps induced by the vomiting of the first acid-matters of the stomach. But although it is generally associated with that stage of the disease, it is not in the relation of cause and effect, for this condition of the tongue has frequently been observed previous to the stage of acid elimination. A modification of the curdled tongue is the *tessellated* tongue. In this case the villi are not tufty and flaky, but appear like separate pavement-shaped forms on the surface of the tongue. This is a more advanced stage of the morbid tongue, but the altered appearance is probably due to a greater shortness of the villi from *wear*. The tongue (where there is no inflammatory complication or head affection) is always sufficiently moist during its changes. The next step in the tongue symptom is the peeling of the epithelium. This generally begins at the point, and proceeds to the edges and down the raphé, and may continue till the whole surface is denuded and the papillæ obliterated, and the tongue becomes smooth and dryish, and the colour and appearance of raw beef. This desquamation of the tongue, as in the fatal case of Juan de Nobriga, may extend into the larynx and bronchi, causing complete aphonia and dry sonorous rhonchi under the stethoscope. It is seldom that the basement membrane is eroded or ulcerated; but this sometimes happens. In the late stages the tongue is liable to be incrusted by the buccal hæmorrhage. In one case, ulceration of the tonsils was observed. One death occurred from gangrene of the larynx, and suffocation therefrom. In the "smouldering" form of yellow fever, and when the case is passing into convalescence, and where there has been little peeling of the epithelium, the fur at the base and centre frequently appears as if stained with tobacco juice. This appearance can seldom be traced to any lesion of the tongue itself, but is found to be a

blood-stain derived from the gums at their line of junction with the teeth, and manifests the hæmorrhagic tendency in its lowest degree. In some protracted cases of recovery, an aphthous condition of the tongue has been observed. But, in general, the raw denuded tongue appears in convalescence as if brushed over with a thin coat of milk and water, and the epithelium and papillæ are speedily restored.

Though the tongue symptoms were so striking and characteristic in our epidemic, they seem either to have been undeveloped or unnoticed in the epidemics of Cayenne and Surinam. The following is all that the medical report of the Council of Health of Cayenne to the Minister of Naval and Colonial Affairs says on the subject:

"Langue. La langue était blanche, muqueuse elle devenait râpeuse quand les hemorrhagies buccal se declaraient. Quand l'enduit blanc-jàunâtre qui la recouvrait devenait visqueux collant, c'était de fort manvais augure; il en était de même lorsqu'elle offrait des colorations diverses et sinueuses lui donnant l'aspect irregulier d'une carte de géographie."

In the report of the health-officer of the first class to the Governor of Surinam, the following is a translation of what is said of the tongue in the epidemic of Dutch Guiana: — "The tongue was generally broad, covered with a dark slime: only in a few cases were the edges more than usually red. In general, the tongue was moist and sticky." In this short statement of the symptoms, it will be perceived that the red appearance of the edges of the tongue had been an object of observation, and its rarity was positive, and not due to inattention of the observer. An explanation of this may perhaps be gathered from a subsequent part of the health-officer's report. He says—"A troublesome sequela of those cases that speedily recovered was the abrasion of the skin around the anus, in consequence of severe purgation. One hundred and sixty-three cases were treated for this." The cause of this excoriation round the anus, assigned by the health officer, does not seem satisfactory. It appears to me that the seat of the capillary irritation in the Surinam cases was displaced, and that the rectal end of the alimentary canal would, if examined, have presented some of the appearances which we expect in the tongue, and that the cæcum and colon took the place of the stomach and duodenum, forming the intestinal variety of yellow fever, a few cases of which were seen by us in the present epidemic. Excoriations of the anus and scrotum were rather rare symptoms with us, and seldom observed except in the last stages, or in convalescence. The tongue symptom is one of the highest value. Yellow fever by it has been detected under its strongest disguises. In the case of a Portuguese, who presented himself at the admission-room, and complaining of his side and cough, the co-existence of pleuro-pneumonia with the epidemic disease was instantly diagnosed by his red and partially peeled tongue.

Herpes labialis was rare, but looked on as a favourable sign when it appeared. Like the other individual symptoms, when it appeared at all, many instances of it came together. The vesicles were not so perfect as in those of intermittent fever, or from solar exposure. They contained less fluid, and finally became bloody crusts, but never had the appearance of rupia. In some cases the scarlatinoid rash caused turgescence as well as efflorescence of the skin, as in the fatal case of Antonio Fernandez,

which did not subside till after the establishment of black vomit, when it subsided. When patients have recently arrived from a cold climate, and have a fine, delicate, sensitive skin, their legs, arms, chest, and all exposed parts, are frequently covered with rose-coloured spots of a somewhat circular shape, varying from the size of a flea-bite to what might be covered by the point of the finger. Some are flat, some a little elevated, and some have vesications. These are mosquito wounds, and become hæmorrhagic at the end of the disease, if it terminates fatally. There is a different kind of exanthem from those already described, which I saw best marked in two young men *ex*-"Livonia" in private lodgings. It consisted of inflamed patches, also chiefly over legs and arms, but there were many over the body also. There were no vesications. The lodgings were very much infested with mosquitos, but after extensive experience in all the variety of mosquito wounds, I would hesitate to ascribe those appearances to that cause. Both cases were of the highest grade of yellow fever, but they recovered. The *stomach kept quiet*, and the recovery was, perhaps, in a great measure due to the diversion of the congestive tendency from the centre to the circumference, set up by this exanthem.

There is another *external* or *surface symptom* connected with the skin. The face, chest, arms, and legs have sometimes a slight purplish appearance after the second or third day, and sometimes the colour of a boiled lobster. This appearance varies much in degree, but may be detected by pressing the hand flat on the chest, when the fingers will for a short time be delineated in white with purple outline. This symptom occurs chiefly in the "smouldering" form of the disease, and is often so deep as to conceal the jaundiced appearance of the skin. It is quite different in its character and nature from the cuticular efflorescence before alluded to. The one is active, the other passive—the one is inflammatory (specific and peculiar), the other congestive; the one apparently from the direct action of the irritant poison, which induces the disease, yellow fever—the other a secondary or tertiary effect. This *languid capillary circulation*, as it is called in the case-books, is generally seen, as has already been remarked, in the "smouldering" form of the disease, and is looked on with favour. It would seem to indicate that the congestions were selecting the periphery of the body for their pressure; or that the vital internal organs were relieved of a part of their load by the hyperæmia of the skin. In these cases the skin is generally cool and moist, and sudamina occasionally appear. Desquamation of the cuticle of the front of chest, and of the hands and arms, is sometimes observed in convalescence. But this can scarcely be considered as having any relation to the scarlatinoid rash. It occurs in sailors while in hospital, no matter what has been their ailment, and is confined to the sun-burned parts of the body. In the advanced stages of yellow fever, the capillaries of the conjunctiva, where the eye has been markedly affected, become coarse and enlarged, and the red injection has become orange, and there is a gumminess of the eyes and lids. A little splash or spot of ecchymosis is also common below the tunic at either angle of the eye. If the stomach has remained quiet, and the secretion of urine has ceased, the pupil it is likely is contracted, the palpebral apertures are narrowed, the brows are a little corrugated, and the light is unpleasant, and there is something of a titanic physiognomy.

This condition was very marked in the case of the captain of the *Hinda* (private lodgings), and also in that of Mr. G. and the Rev. Mr. L. It is generally associated with nervous symptoms, or restlessness, or irritability, or joviality, and is one of the manifestations of uræmic intoxication in yellow fever.

There is no mystery in the yellow suffusion of the skin and eye in this disease. It has over and over again been demonstrated to be occasioned by the presence of bile. The tint is seldom deep, except when jaundice supervenes on convalescence as a sequela. It appears in the primary disease associated with an active condition of the liver, and a full supply of bile in the alvine evacuations. It is one of the earliest signs of those internal irritations and congestions in which most of the viscera begin to become involved after the disease has been of one, two, or three days' duration, and the blending of the yellow and the red in the capillaries communicates obviously the orange tint to the sclerotica. The yellowness of the eye is soonest observed at the angle formed between the eyelid and eyeball, and the lid should be turned down while the patient is directed to look up, in seeking for its early detection. It is valuable as a signal of the attack on the liver in the procession of morbid actions, and as a criterion of considerable accuracy of the degree of lesion or disturbance of that organ. The observation of this symptom, however, is subject to fallacy. There are some sclerotics naturally tinted. This is particularly so with Coolies and Negroes, and the mixed races, in whom a little of the black pigment is frequently found in the sclerotica, giving it a smoky appearance, and which has been mistaken for the bile tint. In such cases the report of yellowness of eye would be premature. An error of an opposite kind consists in overlooking sometimes this symptom when it is actually present, and until the degree of it is so deep as to be noticeable on the exposed parts of the eye and skin. By examining, however, the line of junction between the lids and eyeball, both sources of fallacy will be avoided, for there the earliest trace is to be found, and it is seldom the site of accidental discoloration. The difference of appearance between the eye in advanced stages of yellow fever and the eye of jaundice, consists in the absence in the latter of vascular injection, and the presence of a flat gamboge colour only. If we had a case of mild ophthalmia occurring in a jaundiced patient, then, no doubt, the resemblance would be complete. When there has been little or no irritation of the eye in the first stage of yellow fever, the yellow suffusion is simply jaundice. Cases have occurred in which a notable quantity of bile was detected in the urine before discoloration could be discovered in the eye. And fatal cases have occurred, though rare, in which, till the very day of death, no yellowness of the white tissues nor biliousness of urine existed—because, as revealed by post mortem examination, the liver had not suffered in those instances.

Among the surface symptoms may be placed *Epistaxis*. But this will be noticed when the blood comes to be considered. It sometimes happens early in the disease, and it is then an active hæmorrhage, caused, probably, by the dynamic power of the circulation during febrile excitement. In the late stages of the disease, however, death has supervened from incontrollable epistaxis, and then, probably, it originates in the same patholo-

gieal condition as the next surfaee symptom to be described. Generally, bloody furuneles appear late in the procession of symptoms. Their most common site is on the wrist, over the metaearpal joints of the fingers, along the front of the legs, below the seapula, and over the hip, and in the parotid, and on the forehead and lip. They are generally in elose proximity to the smaller arterial braneh es—viz., the ulnar and radial, anterior tibial, glutæal, intereostal and facial arteries. In the majority of eases, these must rather be eonsidered *sequelæ* than phenomena of the disease proper. But so elose are they on the primary affeetion (as in the fatal eases of Miss N. of the *Belairs*, and Mr. L. M., in whom they were eontemporaneous with black vomit, and in the latter ease on the third day of illness), and as they are sometimes even the eause of death in the progress of yellow fever, from hæmorrhage and disorganizing infiltrations of blood, to separate them from the train of morbid proeesses whieh proeeed direet from yellow fever poisoning, would do violenee to truth for the sake of system. Sometimes these furuneles are very tender, are aeuminated, and inflamed; sometimes they form large abseesses of purulent matter, with a pale or an inflamed surfaee, and this ehiefly when below the seapula, or over the hip. Generally on the legs they are flat, present no inflamed appearanee, but show a flat, purplish vesication, about the size of a split pea or a sixpenee. If you open one of these vesieations, a little watery, curdy sanies will be discharged; and you will believe that that is all, and of no eonsequenee. But if you elip away this vesiele, and wipe the bare eutis, you will pereeive in the eentre of it a eireular perforation, into whieh a probe easily passes, and whieh goes down through the true skin and eellular tissue to the surfaee of the deep faseia or the musele. And if you now squeeze on each side of the vesieation, one or two little dark elots or pellets will start up, and be aeeompanied or followed by a little purulent matter. There is no base or hardness; there seems to be no cyst of any eonsequenee; and the whole affair will elose up and heal, and require no further treatment than the emptying it. Now this is the simplest form of that morbid manifestation. But when it oeeurs over a joint, or below a strong eonfined faseia, abseess, with diffuse phlegmonous inflammation—or in a vaseular tissue, as the parotid gland, death, from destruetive infiltration of blood, gangrene, and hæmorrhage, may follow. The formation of these bloody furuneles is, it is likely, not eonfined to the external parts of the body. In the case of Ballobitch (Seaman's Hospital), the post-mortem examination disclosed a eondition of the kidney whieh was probably due to this eause. The following instanee may be given as an illustration of a ease with bloody furuneles, although they appeared in *convalescence* from a graver attack. It is eited, beeause by it I beeame enlightened as to what, I believe, is the true nature of these (which, for want of a better name, we eall) bloody furuneles.

Peter Daley, of the ship *Alenker*, was attended by me in private lodgings, in January, 1853, and reeovered. The following is an extract from my notes:

"*Bloody Furuncles: considerable loss of Blood on 11th day of illness of Yellow Fever.*—Jan. 27th. Peter Daley, referred to at pages 29 and 31, has lost about eight ounces of blood from a bloody furuncle on wrist of left hand, and another on the metaearpal joint of little finger of right hand, to-day. I have had to apply

compresses to each. That on the wrist began about five days ago like a 'blind boil,' and was tender, for he winced on several occasions when feeling his pulse. The swelling afterwards became distinctly acuminated, and below the cuticle there was lividity, as if some bloody ichor was extravasated there, but not amounting to vesication. At present, having burst two or three days ago, the cuticle seems undermined and separated for about the diameter of three-quarters of an inch, and there is a decided loss of substance, a sinking below it. Out of this occasionally sprouts a small mass, about half the size of a filbert, but elongated, of blood, like the 'bullock's liver' of scurvy; or rather like the clotted blood which escapes when, with a lancet, you divide a recent painful external hæmorrhoid. But when this is removed, florid blood trickles out rapidly, as if an arterial twig had been opened. There seems to be the separation of a small slough this morning, after the poultice was removed (for it and the other had been poulticed till to-day). The furuncle over the finger is also very painful, and the joint swollen. Clots of blood can be squeezed out from below the fascia for the distance of an inch and a half. The edges of the orifice are livid and unhealthy. His urine is still very copious. There is still albumen in it, though not much. He had a tolerably good night last night, though the previous night's rest was bad. The night before that again was good, for I had given him some drops of solution of acetate of morphine (quarter of a grain). If we look on the disease as having passed on the 7th day, these furuncles may be considered as *sequelæ;* but they are sometimes seen in the advanced stages of bad cases. He has many small ones on different parts of his body; but those mentioned only are such as require medical treatment. I prescribed five grains of gallic acid every three hours=four doses. Do these half-active, half-passive hæmorrhages arise from the dissolution of the solids or fluids? I incline to the former.

"Jan. 31st. Peter Daley (page 37) has to-day a large phlegmonous swelling over entire surface of left hip, threatening extensive suppuration. In taking off the bandages used in restraining the bleeding, to-day, the following were the appearances—i.e., over the metacarpal joint of the little finger of right hand, and extending about two inches in long diameter,—the cuticle is elevated and separated from the true skin. When it is removed, an ulcer over the joint, about the size of a sixpence, is discovered, of considerable depth, and showing the plugged mouth of an arterial twig, from which the hæmorrhage must have proceeded. The edges of the ulcer are well-defined and clean, but the base is foul. On the left wrist, over the projection of the ulna, there is a similar ulcer, but cleaner, and with an evident tendency to heal round the edge. This ulcer, like the other, is excavated clean down through the skin and integument. It has no surrounding separation of cuticle, and no plugged vessel that I can see. Indeed, from the furuncle which preceded this ulcer the hæmorrhage was comparatively trifling.

"Feb. 1st. Peter Daley (page 39) was seen again by me about two hours ago. The ulcers over little finger and wrist have almost *completely* filled with healthy granulations since yesterday (twenty-four hours). There was little redness over left glutæi muscles to-day; but feeling distinct fluctuation, I punctured the tumour, and about half a pint of thick altered blood escaped, with a few clots of purulent matter. This sequel of yellow fever is now clear to me. In this case, as, no doubt, in others, the cause of these tumours and furuncles is the *rupture of an arterial twig;* and the presence of the extravasated and decomposed blood sets up a certain amount of irritation or inflammation, which extends to the skin, giving it the flush, and causing the presence of more or less pus in the cavity. These swellings are generally painless, and are discovered, as it were, by accident. I have no doubt that in Peter Daley's hip, the vessel had ruptured two or three days before the ailment was discovered; and if I had made a more careful examination yesterday (when my attention was directed to the hip, from observing the difficulty with which he got up from his bed, and not from any complaint that was made), I should have detected the fluid, and let it escape. When these abscesses (?) are emptied, they heal immediately; they have no inflammatory cysts to be dispersed.

"Feb. 2nd. Still very copious rusty discharge from hip. Vesications on legs drying, but containing blood. Has had to take morphine for restlessness for the last two nights, and, consequently, magnesian mixture to-day.

"Feb. 3rd. To-day, a prominent bloody vesicle has appeared over upper end of right fibula, less than the size of a sixpence. I clipped off the cuticle, and wiped away the altered blood with which it was filled. It appeared, before being cut, to be quite on the surface—cutaneous—with a slight areola; but on examining the part carefully, I observed a circular perforation, which penetrated into and through the cellular tissue, and leading evidently to a cavity. On squeezing this, blood and pus exuded. It seems to me that here, also, an arterial twig has given way, and a little false aneurism had caused the ulceration, suppuration, and vesication.

"Feb. 4th. Peter Daley's abscess (?) over hip has ceased to discharge to-day. Of the little cavity, ulcer, and vesicle over fibula, nothing remains but a blood-stain on the skin. Ulcers of wrist and fingers cicatrizing. He goes on board to-morrow."

Several similar cases, such as those of Major and Anderson, were in the Seaman's Hospital at the same time; and by further observation of them and others subsequently, the conclusions which I had arrived at from Peter Daley's case were confirmed.

But there is another species of abscess which occurs as a sequel of this disease, and which I have never seen in the primary stages; and Peter Daley himself affords an example. In a note of the 30th of January, I have entered thus: "To-day, I detected a slight, livid, painless swelling over his left eyebrow, *which had come since yesterday*, and on opening it, about two drachms of apparently healthy pure pus escaped." Whence came this? was it absorbed from the bandaged ulcer—could it be?

High temperature of the body seems to have persisted longer through the stages of the disease in this epidemic than the past. There is great irregularity in the temperature of the surface. Sometimes the forehead is the hottest part of the body, and occasionally the chest. The uncovered portions of the body in the late stages are easily reduced in temperature; and thus, while the exposed chest and extremities may feel cool to the touch, the axilla may raise the thermometer to 102° or 103°. The highest temperature I have observed in the axilla during the course of the disease was 107°.

We have now considered the chief symptoms ascertainable from the testimony of the patient, and by the observation of the surface of his body. Those depending on an examination of the *secretions, excretions, blood,* and *breath,* will next be considered. And here it may be remarked, that the test-tube and microscope are as necessary for the correct diagnosis and prognosis of yellow fever, as the stethoscope and pleximeter for diseases of the chest.

CHAPTER IV.

At page 93, in the 'Account of the last Yellow Fever Epidemic of British Guiana,' there is a note by Dr. Davy, in which is mentioned the discovery, by Dr. Collings, of albuminosity of urine as a characteristic of yellow fever. This important discovery was duly appreciated in the investigation of the phenomena of the present epidemic. The following remarks will embody the results of observations made on the urine of yellow fever generally, since the 6th of February, 1852. The urine is

always *acid* in the first stage, and continues so generally till convalescence, when it becomes alkaline, or until it becomes heavily charged with bile. In the case of Macey (Seaman's Hospital), the urine in the advanced stages was neutral on being passed, and immediately became intensely alkaline. This happened also in the case of Ellwood (Seaman's Hospital). These instances of alkalinity in fresh urine seemed due entirely to the presence of ammonia. Numerous experiments on the specific gravity of the urine were made without any striking general result being elicited. During the early stage of the fever the urine is normal in colour, clearness, and quantity. As the disease proceeds, about the third day, the colour alters, and becomes that of sulphur, or primrose, or straw, or light gamboge, and is perhaps slightly turbid, with a little floating sediment. The colour, during the progress of the disease, deepens, till it becomes yellow or orange; and if the case end in convalescence, the urine is very copious, and may appear, *en masse*, black. As the colour deepens, the sediment becomes more decided, both in quantity and gravity. It is, however, seldom very considerable in quantity, and might escape careless casual observation. But in one case, that of Theodore Ternaban (Seaman's Hospital), it contained a sediment which occupied one-half the urinal. If the case is going to terminate with *suppression*, it generally does so in an abrupt manner. At other times, when the event is to be the same, the urine is expelled of an amber colour and of an oily consistency, and in quantities of a drachm to an ounce, as if with some tenesmus of the bladder. In one case only was diuresis noticed (Barnet, Seaman's Hospital, March, 1853) during the active course of the disease. This was after the use of a tobacco clyster, and was at last followed by fatal suppression. About the month of June, 1853, the physical appearance of the urine was for a few weeks considerably altered. It then had a pale watery or smoky appearance, with a layer of blood corpuscles as a sediment; and in some cases the urine was *very bloody*. In uncomplicated yellow fever the urine is never buff nor red (unless from blood), and a glance of it at the bed-side of the patient has been sufficient to correct at once erroneous impressions as to the nature of the case. Fevers of rheumatic and inflammatory origin have thus been discriminated from the epidemic. A pinkish sediment was observed in a few intermittent fever complications. Malingering is very rare during an epidemic; indeed, among the seamen, the chief difficulty lay in inducing them to enter the hospital early enough. There was one exception, however, and he was detected at once by the urine. Quillan, on the 9th of February, 1853, returned to the hospital after having been discharged cured of an attack of yellow fever. He complained much of nausea, and showed copious vomit in his basin. Pulse was very quick, and he looked ill and prostrated. On looking at his pot, full of pale, non-coagulable urine, his case was seen through forthwith. He was partly malingering and partly suffering from hysterical excitement. On further inquiry, I found that he came from the *Copia*, the master of which we had had fined for neglect of his sick men, and who had the very worst reputation among the seamen for his severity or brutality. The *Copia* was about to sail, and Quillan's plan for leaving the vessel was to sham sickness. The crisis of his fate rendered him nervous, and his urine betrayed him. The

copiousness and wateriness of hysterical urine contrasts well with that of yellow fever. During the progress of the disease *retention* sometimes occurs from apathy. The patient does not pass it, either because he thinks he cannot, or he feels no impulse. But in such cases, if he is told to do so authoritatively, it will be done. Retention requiring catheterism occurred in six individuals in both hospitals. In one of these (Swede Anderson, Seaman's Hospital) the retention occurred in convalescence, and required three operations. When *suppression* occurs in the course of the disease, it may be regarded as the most fatal sign. In one case, however (Barkway, Seaman's Hospital), the secretion was restored, and the patient recovered. About the period when the urine changes its colour, and particularly if there be turbidity, if in quantity more than three or four ounces, it will, when recently passed, appear *frothy*. It then contains albumen—for ascertaining which the double test of heat and nitric acid should always be used. It is also well to be apprized that urine was observed about the month of June, 1853, in which albumen, though present, did not answer to the double test till the specimen was put aside, and suffered to *cool*. The cases wherein this happened were chiefly dissipated subjects, but not in all instances. Albumen appears on the second or third day generally; but in a few days it has been found as early as the first day of illness; and in a few cases it did not appear till the day of death, and after black vomit had set in. In several instances on the fourth day, when the tongue was completely denuded, the urine was not yet coagulable. Albumen was seldom seen in aborted cases. In a few of these it appeared during their convalescence—for instances, the following may be cited: Peter Kayle, 19th of January, 1853; Henry Russel, 27th of November, 1852; M'Grigor, 19th of October, 1852; John Smith, 29th of October, 1852; Poole, 7th of December, 1852,—all of Seaman's Hospital. During that period of the epidemic when torpidity of the bowels was observed, and croton oil was occasionally required as an early purge, the urine was later in becoming albuminous. In three cases the albuminosity was *intermittent* for one or two days. These were Asthrup (Seaman's Hospital, April, 1852), Profine Martinez (Colonial Hospital, June, 1852), and John Ferguson (Seaman's Hospital, October, 1852). Albumen appeared in every fatal case of normal duration. It sometimes ceased in convalescence suddenly, always before the yellow suffusion of skin and eye, or bile in the urine, disappeared, except in the single case of Manuel de Nobriga, in whom Bright's disease seemed to be a sequel of yellow fever, and who, after remaining in hospital upwards of two months, left with his urine still albuminous. Between the eleventh and twentieth day of gravior cases, it generally disappeared, and its disappearance formed the criterion for the discharge of the patient from the hospital. The colour of the precipitated albumen was never *white*, as it is in our cases of Bright's disease. It is doubtful if the primrose or sulphur colour is due to bile. In several such specimens the nitric acid failed to bring out the bile tint, although the urine was coagulable (as in the cases of Jones and Collard, Seaman's Hospital, December, 1852). But the gamboge yellow and orange colour were clearly referable to that source, as daily experience with Heller's test demonstrated. As before mentioned, bile was thus occasionally detected in the urine before the eye or skin was appreciably discoloured. In one

case (that of Bevan, Seaman's Hospital, 1853) the urine remained abilious till the period of his death.

The *turbidity* of the urine was not necessarily connected with its albuminosity. The urine may be deeply tinted with bile, and highly albuminous, and yet clear. The turbidity of the urine was caused by the presence of mucous epithelial matter, coagulated albumen, and casts of the urinary tubuli. It is probable that the free acid of the urine has a coagulating power, and sometimes communicates turbidity. The presence of mucus will have a similar effect, but the turbidity then is not general, but occupies a lower stratum of the fluid, and is light and floating there, while the supernatant liquid is clear. There is nothing distinctive or of importance in this mucous condition when the urinary secretion is copious. Perfect epithelial scales are rarely found in the sediment, but broken-up epithelial matter is abundant. In the case of Ternaban, before referred to, the appearance of the sediment to the naked eye was that of pus. The microscope showed this enormous mass to be broken-up epithelial matter. It was all soluble in *liquor potassæ*.

There is a variety of urinary sediment which appears of a yellowish-brown colour, a little darker than the fluid in which it is contained, and exists in small curdy-looking masses. It is only partly soluble in liquor potassæ or nitric acid. Under the microscope it has a fibrillated appearance, and it entangles numerous tube-casts and large organic globules and epithelial scales. This variety is rarely seen, but when it is, it bears the most fatal import. There is another variety of the "curdy sediment," in which this particular material is deficient; but it also entangles tube-casts, and it appears to be composed of amorphous epithelial matter, and when treated with acetic acid, shows large and small organic globules—the latter about the size of mucous corpuscles; and these bodies seem to constitute the mass of this curdy sediment. In all cases, except the two now mentioned, the tube-casts roll separately and detached in the urinary sediment. Probably one of these may be the matter which was seen by Dr. Collings, and was considered by him to be of the nature of *casein*. I was impressed with the opinion that fibrine entered into the composition of the curdy sediment, and also that the same material constituted the basement membrane of the tube-casts.

Although albuminosity is almost always the antecedent to the presence of tube-casts, a case (that of William Narro, November, 1852, Seaman's Hospital) occurred in which they were found in non-albuminous urine. The tube-casts are generally short, thick, and club-shaped, and nearly opaque. Along with them, also, there are frequently amorphous bodies, apparently of the same material. The casts consist of a basement membrane, and are covered with minute pavement epithelial scales, between the interstices of which there is some translucency. At one end they have generally a broken-off appearance, but some are round at both ends. Few are equal in diameter throughout their whole length; some are sacculated, some fusiform. They are sometimes slightly stained with hæmatosine or bile. At the broken end they are frequently destitute of epithelial covering. A long tortuous cast is occasionally seen, but being nearly transparent and without epithelium, may escape observation. I measured three specimens of tube-casts from Chugg and Holmes (Seaman's

Hospital, 4th of December, 1852), and Feliciana de Jesus (in Colonial Hospital, 8th of December, 1852). The measurements were made with Ross's micrometer eye-piece, and show the greatest length and breadth of each cast observed, in parts of an inch. Chugg's were $\frac{1}{210} \times \frac{1}{950}$, $\frac{1}{270} \times \frac{1}{950}$, $\frac{1}{633} \times \frac{1}{950}$, $\frac{1}{270} \times \frac{1}{380}$, $\frac{1}{210} \times \frac{1}{633}$, $\frac{1}{158} \times \frac{1}{475}$, $\frac{1}{126} \times \frac{1}{475}$, $\frac{1}{210} \times \frac{1}{661}$, $\frac{1}{190} \times \frac{1}{1266}$, $\frac{1}{633} \times \frac{1}{950}$, $\frac{1}{633} \times \frac{1}{950}$, $\frac{1}{190} \times \frac{1}{661}$. Holmes's were $\frac{1}{146} \times \frac{1}{950}$, $\frac{1}{950} \times \frac{1}{1266}$, $\frac{1}{380} \times \frac{1}{1266}$, $\frac{1}{316} \times \frac{1}{950}$, $\frac{1}{146} \times \frac{1}{1266}$. Feliciana de Jesus' were $\frac{1}{190} \times \frac{1}{475}$, $\frac{1}{360} \times \frac{1}{475}$, $\frac{1}{156} \times \frac{1}{422}$, $\frac{1}{211} \times \frac{1}{633}$. As tube-casts are so soluble, not only in liquor potassæ, but also in ammonia (though more slowly), it is necessary to look for them before decomposition of the urine takes place. The scanty, acid, amber-coloured urine, of oily consistency, in the last stages of yellow fever, is always highly coagulable, but contains no tube casts, and is loaded with mucous corpuscles.

It is very common, before the urine becomes albuminous, in using the nitric acid test, to perceive considerable effervescence, although the urine be acid, and no carbonate of ammonia can be suspected as being present. It is likely due to the decomposition of uric acid, or urea, by nitric acid, while the urine is heated. Is the effervescence in such cases due to an excess of one or other of these; or is it a normal condition, and are the instances of non-effervescence due to the deficiency of either? Whenever urine has been set up and examined for uric acid, it has always been found. But, except in the case of Profine Martinez, before referred to, I recollect of no instance in which it has been found as a sediment. In Martinez' case, it was on the tenth day of his illness on which it was observed, and the deposit was copious. In the case of Morgan (Seaman's Hospital, September, 1852), his post-mortem urine was examined both for urea and uric acid. The hydrochloric acid test discovered the latter, but no urea could be detected, although, in the single opportunity for experiment which occurred during life, it was found to be copious. The specific gravity of the urine during life was 1·023 at 85°. The sediment was loaded with tube-casts. When heated, the coagulum occupied more than a quarter of the space in the tube. When the albumen was separated by filtration, the urine yielded nitrate of urea so copiously, that it became solid. Yet the urine found in his bladder after death did not yield a trace. On the same day that Morgan's urine was examined for urea, Gilmey's, also, was tested for the same. The coagulum of his urine occupied upwards of a third of the space in the tube, and about one half of the specimen tried for nitrate of urea became solid: it appearing, from these two experiments, as if the quantity of urea present was in inverse ratio to the albumen. In two or three experiments for phosphoric acid, it was found abundant in yellow-fever urine.

The observer of this description of urine cannot but be struck by the rarity of the presence of crystalline bodies in it. After a time he ceases to expect them. There were only six cases in which, during the active course of the disease, triple phosphates were found. There was one case in which a copious sediment of urate of ammonia was present, with the urine still acid. This was in one of the two cases of the "Livonia" before referred to, in which the red patches appeared on the skin. After convalescence, the albumen was generally replaced by the earthy salts and

triple phosphates and urate of ammonia, and the tribasic triple phosphates were frequently seen. On the 4th of December, 1852, however, while examining the urine of Chugg, before referred to, I discovered distinct and well defined minute octahedra of oxalate of lime in the sediment, which also contained numerous casts of tubes, epithelium, and apparently coagulated albumen. Associated with these were vibriones and moving monads. About the same time, in W. Bertie's urine, I discovered oxalate of lime crystals and vibriones. The urine on both occasions had stood for twenty-four hours. As yellow-fever urine is such as, *à priori*, oxalate of lime might be expected in, I surmised that it had previously escaped our observation by our neglect of the rules given for detaching it, and by observing the urine too soon after emission. Some of Robert Forsyth's urine was, consequently, put by the same day for examination for this salt of lime. After remaining twenty-four hours, the following were the results of observation: "Coagulable; numerous casts of tubes, most of them perfect, of average size, a little yellowish in colour. With the one-eighth inch object-glass I can detect no animalcules nor crystals. It is still slightly acid." Twenty-four hours afterwards, the phial having, in the mean time, been kept firmly corked, "sediment loaded with well-defined triple phosphates; two quartz-like uric acid crystals under the glass, slightly claret-tinted. The casts have nearly disappeared. The two in view are thin and wasted, but still retaining the yellow tint. The urine is now strongly alkaline." Subsequently to this experiment, I set aside seven different samples of yellow-fever urine for the purpose of examining it for oxalate of lime, under the guidance of Dr. Golding Bird's directions. The first four numbered specimens remained well corked and undisturbed fifty hours, for the deposition of crystals. The last three remained twenty-eight hours. Each specimen was first examined by a half-inch, and subsequently, by a $\frac{1}{8}$th inch object-glass of Ross. 1st. "Alexander Muschard.—Urine still acid; pellicle of fungus on surface; a few tube-casts observed, and much comminuted matter, probably of the same material; numerous sporules; no crystals; no vibriones. 2nd. George Thompson.—Urine still acid; several organic globules; much epithelial matter; a very few casts of tubes, tinged yellow; sediment copious but floeculent, and floating; no crystals, no vibriones. 3rd. Michael Flynn.—Sediment heavy; urine fetid; slightly alkaline; four crystals of triple phosphates in drop under observation; a few small casts of tubes; numerous amorphous pieces, probably of the same material; numerous luminous little spheres which, under a $\frac{1}{8}$th inch glass appear to be oval sporules; some vibriones. 4th. Antonio Ballabatch.—Urine still acid; sediment dense; very numerous tube-casts, faintly yellow; three of them in drop under observation deeply blood-tinted; some blue pieces, possibly extraneous; a few organic globules; minute luminous points, which under high power prove to be vibriones; no crystals. 5th. Michael Flynn (same patient as gave number 3).—Urine fetid; sediment heavy; still acid; numerous casts; amorphous pieces; two large casts, deeply-coloured orange; one amorphous piece, the same; several straight transparent casts without epithelial covering; many comminuted pieces of yellow tint; no crystals; vibriones seen by high power. 6th. Robert Forsyth.—Urine alkaline; copious buff sediment,

part of which adheres to sides of phial; no casts; sediment consists of triple phosphates and urate of ammonia; no oxalates. 7th. Adam Smith.—Urine neutral or slightly alkaline, of vinous-urinous odour; sediment half dense; triple phosphates very numerous; casts of tubes becoming thin; no oxalates." Vibriones were seen on several occasions in fresh urine; but all in the most violent cases. For instance, in the case of Bruce (Seaman's Hospital, October, 1852), three hours after the emission of the urine; and in the recovered case of the master of the *Margaret Poynter* (private lodgings), after the occurrence of black vomit, on the fifth day of his illness, in a peculiarly dark sediment of fresh urine.

On the 17th of May, 1852, the first sporadic case of *bloody urine* was noticed. A year afterwards, it became a symptom of frequent occurrence, grouping, and giving a character to the cases, and then disappearing, as has been already noticed. In a few cases, such as that of Johnston (Seaman's Hospital, October, 1852), it assumed the form of active hæmorrhage. It appeared on the first day of fever in the case of Farish (Seaman's Hospital, June, 1853). In many of these cases of bloody urine there were no casts of tubes or of epithelium; and in a few cases there was "smoky" urine, with a thin layer of blood corpuscles as a sediment when the supernatant fluid showed only a mere trace of albumen (as in the boy Alger, Seaman's Hospital, 5th of January, 1853). In some few cases the blood intermitted, as in the cases of King and M'Call (Seaman's Hospital, January, 1853). In the latter case, the bloody urine of the morning was succeeded by pale urine, with cloudy sediment, which consisted of mucous corpuscles and organic globules, in the evening. The bloody urine, in many cases, seemed a favourable sign; and the interpretation of it probably was, that the hæmorrhage, proceeding from the calyces or pelvis of the kidneys, tended to relieve the congestion of the secreting apparatus—such as in the interesting case of King, above referred to, wherein the urine was bloody, highly bilious, and copious.

These observations on the urine of yellow fever, refer chiefly to that of males. With that of females, the difficulty of obtaining pure specimens was almost insurmountable. This arose, not only from the action of the bowels, but at that stage when the urine should become a study of great value and interest, the catamenia were sure to appear, whether due or not, and thus effectually embarrass the examination of the urine. My impression however is, from the few imperfect observations that could be obtained, that the urine is found less frequently, and less highly, albuminous than in males; that it is more quickly and abruptly terminated in convalescence; and that the flow of urine is fuller throughout. If the numerical method of induction applied to this subject should confirm this opinion, what is the rationale of the fact? Is the female urinary apparatus better endowed than that of the male? Is there any relation between the tendency to suppression and the calibre of the tubuli uriniferi? Or does the vaginal or catamenial hæmorrhage tend to relieve the renal congestion?

While our experiments and observations on the urinary symptoms were going on, they were extended to such cases of intermittent fever as presented themselves. In about twenty cases of this disease, contempo-

raneous with the epidemic, the urine was examined for albumen, and in no instance was it found present. One of these cases was that of Josefa de Susa, who had been previously in the hospital with yellow fever. She had had copious black vomit; a bloody furuncle then appeared on the cheek, and terminated by an abscess of the left parotid gland. A singular circumstance occurred in this case. The operation of opening the abscess caused nausea and vomiting, and renewed, for a day during convalescence, the stage of acid elimination. This woman returned to the hospital about two months afterwards, suffering with intermittent fever, and her urine was examined for albumen without detecting a trace.

On the 3rd of May, a mixed case of yellow fever and small-pox occurred in a Portuguese boy, named Manuel Gomes. He had been treated in town by an experienced practitioner for yellow fever, and sent to the hospital, in which the treatment was continued. He had the red-tipped and red-edged tongue on admission, irritability of stomach, with greenish, acid ejections, flushed face, and *albuminous urine*. He had been ill three days before admission, and the day after his admission the first rash of small-pox eruption came out; and subsequently he was removed to the small-pox branch, where he recovered in due time. This case brought attention to the necessity of ascertaining the state of the urine in small-pox, in order to estimate the value of albuminosity of urine in differential diagnosis. Several experiments were then made on the urine of small-pox patients, but no albumen was found. No record was kept of these extemporaneous experiments. But on the 10th of May, all the patients in the small-pox wards were subjected to examination with the following results:

Name.	Number of days ill.	Condition of urine.	Specific Gravity.
— Cuffy.....	42 ...	Noncoagulable ...	1·014
Henry Cumings	14 ...	do. ...	1·012
Tom. Manning	22 ...	do. ...	1·016
Manuel Gomes (above)	8 ...	do. (effervesces) ...	too scanty for urinometer.
George Warren.....	8 ...	do. ...	1·008
Zacharias	8 ...	do. ...	too scanty.
Maria Lewis	14 ...	do. ...	1·017
Elizabeth	22 ...	do. ...	1·008
Diana Sam	10 ...	do. ...	1·008
Eliza Grant	8 ...	do. ...	1·022
Juliana di Silva	12 ...	do. ...	1·023
Susan Ward, no urine at the time of ob.			
Stephen (negro, 2 years old) eruption just out on third day.....		do.	
Mary M'Crae, admitted yesterday, eruption just appearing		do.	

At the end of April, 1853, a Portuguese child was admitted with purpura. She recovered, without medication, in about two weeks. She had no fever, nor irritation of mucous membranes, nor hæmorrhage; but her skin was thickly studded with petechiæ. This is a very rare disease in Demerara. I have no recollection of more than three cases within my experience, and this is the only one which had occurred during several years. The urine was albuminous in this case.

While on the subject of the urinary symptoms, it may be remarked that the report of the members of the Council of Health of Cayenne to the Minister of Marine, on the subject of the epidemic of French Guiana, announces that they carefully examined the urine, and discovered in it and the kidneys a notable quantity of *pus*. The following is an extract from the report:

"Reins.—Les désordres des organes urinaires ont été constant. Les reins avaient perdu leur consistance; on distinguait cependant les deux substances, et la corticale semblait plus molle; toutes deux étaient décolorées; ces organes étaient souvent gorgés d'un sang diffluent et contenaient un fluide lactescent ressemblant a du pus, et même du pus chez un grand nombre de sujets. Un fait digne de remarque, c'est la promptitude avec laquelle le pus disparaissait quelques secondes apres l'incision de cet organe: il faut observer très attentivement pour constater sa presence, au moment de l'incision, car il se confond immédiatement avec les autres liquides." . . "Les urines avaient le plus souvent un teint jaune verdâtre qui leur était communiqué par la bile, de la presence de laquelle nous nous sommes assurés au moyen du réactif signalé par M. Dutrouleau. Elles contenaient aussi du pus qu'il fallait de grandes précautions pour apercevoir, car il se mêlait a l'urine avec la plus grande facilité. Venait-on le filtrer, il restait sur le papier, et alors, au moyen de l'acide nitrique et de l'ammoniaque, on en constatait facilement la nature."

Notwithstanding the great precautions taken, this statement as to the existence of pus in the kidneys and urine, is clearly founded on fallacious observation; and the error is due, no doubt, to the want of microscopic aid in the investigation. If a section of a yellow-fever kidney be made, and a papilla be uncovered from its calyx, and, either with the back of your knife, or finger and thumb, you compress this papilla, a purulent looking drop will be expelled, which under the microscope will show a multitude of epithelial granules, and some more or less perfect tube-casts. Now, undoubtedly, this was the fluid which the Cayenne observers mistook for pus. The extract given was from the chapter on the "*kidneys*." The following is the entire chapter devoted to the subject of the urine, and it will be seen from it, that they were not aware of the albuminosity of the urine in yellow fever nor the nature of its sediment, and how easily, therefore, they fell into the mistake:—"Urines.—La suppression complète des urines ne s'est montrée que rarement et seulement au début de l'épidémie. Quand il y avait suspension on pouvait le plus souvent, a l'aide du cathétérisme, en évacuer une notable quantité. Elles étaient peu colorées ou légèrement teintes de jaune-verdâtre par la bile, quelque fois sedimenteuses. Chez les convalescents, elles devenaient parfois abondantes et colorées."

It is almost superfluous to remark on the important indications that arise out of the study of the urine symptoms in the medical management of yellow fever. Placed in a ward of such patients, and led to the expectation of seeing no other kind of case presented to you for treatment, yet a glance at the urine at the bed-side of the patient may enable you to decide at once that the case in hand is *not yellow fever*. Such instances have happened with the fevers of peritonitis, rheumatism, and pneumonia. Not only are these symptoms extensively useful in clinical diagnosis, but they will probably be found the characteristic of the specific differences of tropical fevers, and may transfer yellow fever to an entirely new place in

the classification of disease. The importance of these symptoms, as an auxiliary to defective *surface* symptoms, in identifying the true nature of the ailment, is at once apparent. The albuminosity, also, furnishes one of the most obvious manifestations of the disease entering its second stage, and its extension to the great solid viscera. Cases have died, as will be explained, even when the urine was full and free; but (as in the case of the mate of the *Sobraon*) life is prolonged thereby; and no guarantee of safety in one epidemic was so sure as an unobstructed action of the kidneys; and no sign, not even black vomit, so dooming as a suppression of urine. Hope then was gone. No matter how desperate the condition otherwise, if there was copious transparent urine, though ever so coagulable, and black as ink from bile, the struggle was hopefully maintained. For it was felt that the system was still competent to the elimination or decomposition of the yellow fever poison. But *suppression* after the abundant appearance, or curdy appearance of albumen and tube-casts, rendered despair reasonable. The scanty oily-looking urine was generally present in cases that might be abandoned. The tube-casts had disappeared—for the capability *of washing out these plugs of the urinary tubules* no longer existed: they were irrecoverably choked; and the bulk of the scanty secretion seemed to be derived, not from the kidney, but the bladder itself. It may be safely affirmed, that for a correct knowledge of the progress, diagnosis, and prognosis of yellow fever, the close observation of the condition of the urine is indispensable; and that after the attempt to abort the disease has failed, the prime object of solicitude and of treatment is the function of the kidney.

CHAPTER V.

When the attack commences, as it frequently does (not sporadically, but in clusters, as is the mode of appearance, disappearance, and re-appearance of the several prominent symptoms) by diarrhœa or cholera, it is seldom that any opportunity offers of examining the alvine evacuations; for this affection is of short duration. When the disease has not so commenced, the first stools observed are generally those resulting from the administration of medicine. In the hospital reports of our early cases, the following phrase frequently occurs:—"Stools characteristic of the powder." These were the evacuations which followed the resolvent or aborting dose and castor oil. They were generally copious, feculent, pultaceous, with old fæces, and sometimes horribly fetid. They had generally a mottled heterogeneous appearance, made up of old fæces and pulpy carbonaceous looking matter, and a copious intermixture of yellow bile with a white *materies*, the latter having the appearance of chopped half-boiled eggs. This was the stool characteristic of the powder. It had brought away old accumulations of constipated matter: it had acted powerfully on the liver and mucous glands, and it had removed the black material which we often see in the early stage of the yellow fever, and which in some cases constituted the entire mass of fæces, and which we named the *melanotic stool*. The cases of Mether, Farry, and Goodnight, in the Seaman's Hospital, about the 4th of April, 1852, furnished perfect specimens of this description of alvine evacuation. The melanotic stool, or patches of it, seems

the first tangible morbid product of the disease, and is highly diagnostic in the first stage. Its tint varies. It is black (as after the ingestion of preparations of iron) or blackish-brown, or blackish-grey. It is always in considerable quantity and pultaceous. Neither in appearance, or in the stage in which it is found, need it be confounded with another dark stool —the scanty *black-vomit-stool*, which appears at the close of the disease. Sometimes, however, scybalæ of the melanotic stool unexpectedly appear in the evacuation after it might have been supposed that the bowels had been completely emptied. Very dark green bilious stool, *en masse*, has been hurriedly mistaken for this; but the former is thin, while the latter is consistent, and tilting up the former against the white sides of the pot readily detects its true colour. It is probable that this melanotic stool derives its appearance from the extravasations of blood in small quantity into the intestinal canal from the cæcum or colon, and this acted on and blackened by some of the intestinal gases, or acids, mixes with the fæces and communicates the characteristic tint. As an indication, its importance may be ranked with that of epistaxis, and below that of the slight florid streak of blood which is occasionally seen with the mucus in the early vomitings, particularly when the quantity vomited is scanty, and there is much retching. The condition of the intestines in which the melanotic stool appears may be the diminutive of that in which a hæmorrhagic dysentery ushers in the fever, as in the rare cases of Lynch (Seaman's Hospital, 24th of December, 1852); or Carmichael (Seaman's Hospital, 13th of February, 1853); or Morris (Seaman's Hospital, 3d of June, 1852), in the fatal cases of which the cæcum presented the appearances generally observed in the stomach.

The appearance of the bilious element of the stools is at first yellow, and subsequently (contemporaneously with the stage of acid elimination) green. Up to the period when it ceases altogether it is generally copious. About the stage when the suppression of urine occurs, the stool becomes abilious, though sometimes a tint of it is observed to the last. After the melanotic stool has passed away, another appears, which is also very characteristic. It is named in the hospital records the "*caddy stool*," from its resemblance to the fine dark sandy mud, so common in our alluvial deposits, and known by that local designation. This alvine evacuation is of a dirty grey colour, abilious, and liquid, with a sediment (caddy like). It cannot be mistaken for the melanotic stool. It sometimes disappears, or is replaced by the restored secretion of bile, as in the case of George Brasset (Seaman's Hospital, 15th of July, 1852), and its sedimentary character as well as its colour is then lost; and when it appears, as in the same case, it is deficient in its crystalline constituents. A small quantity of bile may be occasionally present without destruction of its identity, as in the case of Thomas Young (Seaman's Hospital, 1st of September, 1852). The composition, as well as the appearance of the stool, is peculiar. If a small portion of the sediment be taken up with a pipette, and submitted to the microscope, well defined crystals of triple phosphates and uric acid will be found, sometimes singly, and sometimes together in the same specimen, as in the following cases:—George Brasset and Thomas Young, before-mentioned, J. Doherty (Seaman's Hospital, 20th of July, 1852), Morgan (Seaman's Hospital, 17th of September, 1852), W. Munro

(Seaman's Hospital, 29th of November, 1852), W. Burns (Seaman's Hospital, 26th of December, 1852), and Abraham Limisson (Seaman's Hospital, 25th July, 1852). In addition to the above crystalline bodies, it contains numerous little amorphous masses of black opaque matter, which seems to be its *constant* ingredient. In Brasset's case the uric acid crystals were very numerous in pale cubes; there were also coherent rhomboids, and many small masses like yellow quartz. They entirely disappeared when the bile re-appeared. In Thomas Young's case, when some bile was present, triple phosphates alone were found. In Morgan's case the triple phosphates were absent, and the uric acid was chiefly in long rhomboidal prisms in coherent parcels, and single hourglass-shaped crystals. In the case of Wallace (Seaman's Hospital, 22nd of October, 1852), when a little bile tinged the caddy stool, triple phosphates alone were present. In the case of James Carson (Seaman's Hospital, 24th of December, 1852), some crystalline bodies were observed, the nature of which we could not determine. They were not unlike broken quinine crystals, but they wanted the fibrous character of that substance, and they were much more regular in their outline, sides, and angles, than is ever seen in the sulphate of quinine. They also dissolved in dilute hydrochloric acid, which sulphate of quinine does not, and for the same reason they were not uric acid. They were insoluble in liquor potassæ and ammonia, and as several of them showed some imperfect prismatic forms, I was inclined to believe them to be triple phosphates, although this opinion is discountenanced by the fact that triple phosphates are always so decisive in their forms, and are soluble in the acids mentioned. Among the crystals and black amorphous bodies of the caddy stool sediment evident under the microscope, are also found bright yellow oblong bodies (cholesterine?) somewhat darkened, but not much altered by hydrochloric acid. Their bright yellow colour, unseen by the naked eye, contrasts strongly with the grey and crystalline heterogeneous matter which surrounds them. If the patient had been taking soup, vegetable and other débris (such as the cellular tissue of onions), will be found also in the sediment. This caddy stool may exist without the presence of the urinary crystals. This variety of it was particularly noticed in January, 1853, when blood corpuscles began to appear in the urine, and when it was paler and less albuminous, and the tube-casts were thinner and fewer, and the urinary organs seemed altogether less embarrassed in their functions than usual. At this time also a tendency to torpor was observed in the early stage of the disease, and croton oil had to be substituted for the usual mild purgative. There is a spurious caddy stool which I have observed in the case of Judge (Seaman's Hospital, 24th of August, 1852). In this case the sediment consisted of undigested starch globules, stained by dark green bile, and also bile globules or cells (?) of a striated appearance and bright yellow colour. The caddy stool was observed as well among the Portuguese immigrants as the seamen, and its composition was similar.

As the disease still further advanced, and towards its fatal termination, the alvine evacuations again changed their character, and became scanty and mucous. The mucosity varied much in consistency—from gelatinous, as in the case of H. Collins (Seaman's Hospital, 4th of December, 1852) to that of rice water, as in the case of H. Britton (Seaman's

Hospital, 1st of January, 1853). Its ordinary consistence and colour was that of thick mucilage, and it was more or less in quantity as it was more or less thin. The mucous stool in the case of Laman (Seaman's Hospital, 20th of September, 1852) was half an ounce in quantity; that of Dryburgh (Seaman's Hospital, 25th of July, 1852), about one ounce; that of H. Russel (semi-gelatinous), about one drachm. The mucous stool was also variously tinted. It was sometimes grass, or olive, or spinage green, sometimes fawn-coloured, sometimes primrose, sometimes rusty, and sometimes *brown*, or *black*, or *streaked*. The last described colours were denominated "*B. V.* (black vomit) *stool*." These mucous stools almost always appeared *after* black vomit, and were contemporaneous with the scanty urine before described, when it appears. The alvine evacuations in yellow fever, from the beginning to the end of the attack, are always alkaline, except in one instance—that of the black vomit stool: in that it is always acid. Its chemical quality is evidently due to the admixture of a portion of the black vomit, which has descended (if not found in the intestines) by peristaltic motion into the intestines, and mixed with the scanty mucous stool, and in such quantity as not only to neutralize it, but be in excess. The scanty thick mucous stool—almost a jelly—has generally a little thin serum around it in the bottom of the pot. The bulk of all these varieties of the scanty mucous stool consists of mucus, broken-up epithelial matter, and myriads of epithelial granules. Sometimes little wavy flakes, like morsels of cuticle, are also to be found. They also frequently contain the crystalline bodies of the caddy stool, particularly when they are rather thin and serous. By appearance, they would be taken for rectal stools and the results of tenesmus; but such is not the case. A burning sensation is often complained of, but seldom any tenesmus, and no doubt these stools consist of that mucous matter which we find after death lining the intestinal canal generally. In a few cases, where there has been total suppression of urine, these stools have become diarrhœal, as in the case of the master of the *Hinda* (private lodgings). In him the procession of the symptoms was as follows: "Caddy stool, then a urinous-looking stool without bile, then a reddish mucous fluid, as if altered blood had stained it, then a black molasses-like stool, evidently the admixture of imperfectly formed black vomit,—all within twenty-four hours, during which time there has been no secretion of urine, and he has been very frequently on the chair for diarrhœa. He is becoming very restless; tremor of arms; speech faltering; intelligence dull." He died on the following day. The evacuation of these stools seemed to be a well-meant effort of nature, and compensatory of the suppression of urine. As will be seen by the foregoing extract from my notes, there are several modifications of the *mucous stool*. But all the elementary forms, I believe, are included in the descriptions already given. Sometimes fatal cases terminate with hæmorrhage from the bowels. In such instances, of course, the alvine evacuations just described will not be apparent.

In observing these evacuations, a minute portion placed on the glass slip should always be diluted with a drop of pure water. Great care was taken to prevent fallacy of observation, and to assign appearances to their proper causes. At first we suspected that the mucous stool might

have originated from the irritation of the resolvent dose on the mucous crypts of the intestine, and the more particularly as in some of these stools we detected a few spiculæ of quinine. But we found that cases which had been neglected and untreated, and brought in to us in the last stage of disease, presented the same symptoms. It was also irrespective of the number of doses given, and it was separated by intervening phenomena from the supposed exciting cause. We were hence compelled to infer that this mucous stool at the close of yellow fever forms a portion of the normal morbid phenomena. We also suspected, at one time, that the crystalline matter of the caddy stool might have been due to the actual presence or the chemical agency of the medicinal substances employed; and the magnesian mixture first came under suspicion. But we found the same triple phosphates where this medicine had not been employed. We experimented next on the nitrate of potash, carbonate of soda, and aq. acet. ammon., without success in explaining the presence of urinary salts in the stool. The urine which had been passed, in a different vessel simultaneously with the stool, was also in all cases explored, and care was taken that not a drop of the two liquids had mixed; and in no case were these crystalline bodies found simultaneously in the stool and urine. The minute particles of undissolved quinine certainly resemble uric acid crystals, and when not broken, might, to a prurient imagination, assume the form of triple phosphates. But when large, their *fibrous* structure is apparent, and when broken, the irregularity of their sides and angles cannot simulate regular crystals; and they dissolve in hot alcohol, and while dissolving, show the long striæ of their structure. Feeling, however, our great liability to error in these observations, I transmitted to England a specimen of the *caddy stool* for Dr. Davy's examination. It unfortunately decomposed before its arrival, and all trace of crystalline material had disappeared. It is with diffidence, therefore, that these observations on the composition of the alvine evacuations in yellow fever are offered to the profession. They require the confirmation of future observers. But in the meantime, it would seem as if the intestines could, to some extent, assume a vicarious function with the kidney in yellow fever. *Live* lumbricoides were occasionally vomited and passed by stool during the course of the disease, and found in the intestines post-mortem. In the fatal case of Cornice, of the *Una* (private lodgings), a *dead* tapeworm was passed after the first dose of medicine. In convalescence, after the alvine evacuations have obtained bulk and consistency, they are for some time of a lead colour and abilious, while the urine is copious and charged with bile, and the skin jaundiced. This condition of the stool often changes suddenly. The convalescent vomits up, or passes off by the bowels, a quantity of yellow bile, and the jaundice symptoms begin from that moment to disappear. This secondary jaundice, a true sequel of yellow fever, I am inclined to think is due to ecchymosis around the orifice of the *ductus communis choledochus* mechanically obstructing the vent into the intestines; and its sudden removal arises from the absorption of that ecchymosis and the pressure of the engorged gall-ducts from any sudden muscular exertion.

CHAPTER VI.

The first ejections from the stomach of a yellow fever patient are seldom seen by the physician, but are described to him as food, &c., in a more or less digested state. After these have been discharged, mucus and bile next appear, occasionally with a streak or speck of blood, and with violent retching. The ejections of the stomach are at this time alkaline. The master of the *Valiant* vomited as well as purged yellow bile by pints, up to the fourth day of his illness. This bilious vomiting was succeeded by the ejection of bloody mucus from the stomach, and simultaneously the urine became albuminous. But still he recovered without the case proceeding even to acid elimination. This, however, is a rare case, and generally after the first vomiting the stomach becomes tolerably settled until the second stage sets in, on the second, third, fourth, or as late as the fifth day of the disease. Then, without warning or nausea, the stomach, on any trifling provocation, may eject a quantity of clear, pale, almost limpid or slightly opalescent *acid* fluid, as in the cases of Peter Brodie (Seaman's Hospital, August, 1852) and Harrington (Seaman's Hospital, 7th of December, 1852). Here the disease may terminate or go on to a protracted period, and still make no further progress, the symptom, as it were, becoming a chronic affection, as in the case of Tolloway (Seaman's Hospital); or, as usually happens, this symptom is merely the precursor of a higher and more complex elaboration of the stomach. The symptom now under consideration is the *white vomit*, and indicates the beginning of the stage of acid elimination, and is generally contemporaneous with the first peeling of the tongue. In a few cases, *hoarseness* has immediately followed the ejection of this fluid, as in the case of Mrs. W. But this can scarcely be ascribed to any corrosiveness of the fluid, but rather that in these unusual cases the peeling of the epithelium had been earlier, and extended further into the fauces than usual. In some cases, as those of Mr. W. and Master J. B., this vomit seemed equivalent to the perspiration of intermittent fever, and the whole ailment instantly vanished. In others, as Mrs. H.'s servant, the whole *febrile heat* and excitement ceased, but the disease passed on to a fatal termination notwithstanding, and occasionally the advent of this symptom seemed void of all modifying influence on the progress of the disease. The name *white vomit* may be objectionable as a term to indicate what is meant by it, for this ejection is often bile tinted, but the bile is evidently an extraneous and accidental ingredient in its composition. There is a *spurious white vomit*, which seems to have no critical effect, and is innocuous. It is plentiful, thick, ropy, and alkaline, and consists almost altogether of mucus. Occasionally in true white vomit an admixture with this ropy fluid takes place, and in such abundance as to neutralize the acidity of the former (as in the case of Miller, Seaman's Hospital, 4th of March, 1852), and the presence of the acid is ascertained only by its action on the bile which may be present, and to which it communicates a grass or verdigris green colour. True white vomit is serum, more or less acid, which, after repeated trials, remained clear on the application of heat and nitric acid. Sometimes the stage of acid elimination is first manifested by the alvine evacuations, as in Mrs. H.'s servant, before referred to, and is indicated

by the changed colour of the bile. In some rapid cases, such as that of Mr. Dods (of the *Grafton*, private lodgings), in which the urine was albuminous on the first day, there was no intermediate white vomit or green-tinted vomit. He ejected yellow bile copiously up to the second day, when black vomit came on abruptly. The transition of symptoms, however, is generally gradual, and the white vomit, stained or unstained, is formed, and presents the "snuff-like" specks, the "suspicious sediment," the "*black vomit incipiens*," before it merges into well-defined black vomit. Although there is much reason to believe that the acids of this fluid are those only which are natural to the gastric juices, yet in several cases we have found in this stage the saline acid, as in Ringham (Seaman's Hospital, 2nd of March, 1852) and the same in A. Morison (Seaman's Hospital, 1st of March, 1852) and in O'Donoghue. In the last-named, also, rusty black vomit-looking matter, and highly acid, was expectorated from the lungs and bronchi, as if the acid were a *materies morbi*, or as if other surfaces might assume, under certain circumstances, vicariously, the functions of the great acid secreting organ, the stomach. In the case of Robert Smith, also (Seaman's Hospital, 10th of June, 1853), who suffered from pleuro-pneumonia as a complication of yellow fever, after the *rusty* expectoration ceased, the sputa were of a grass-green colour, apparently from the action of acid on the bilious constituent of the expectoration, while the skin was yellow. The *stage of acid elimination* continues to the close of the disease, and is most intensely manifested during the production of black vomit. Several attempts were made by us to determine the chemical characters of this acid, but without conclusive or satisfactory results. As in the investigation of the alvine evacuations, a difficulty is met with at the threshold, in discriminating what is precisely normal to the subject of examination from what is extraneous and accidental. On one occasion we distilled some of the filtered serosity of black vomit; an acid came over, and we continued the distillation till the vapour ceased to affect the test-paper. The residue in the retort remained acid. Here, then, one point seemed determined—that in black vomit there existed at least two acids, one volatile, the other fixed. We neutralized the distilled fluid with ammonia, and afterwards evaporated almost to dryness, then treating this nearly dry residue with a drop of concentrated sulphuric acid, we detected the *acetic acid* odour. Here appeared another discovery—the volatile acid was certainly acetic. But almost all the patients drink wine. Here was probably the source of this acid. On the 25th of June, 1852, some of the black vomit of Leonard (Seaman's Hospital) was filtered through paper, and put away for future experiment. It was highly acid. After remaining four days it was again examined, and still acid. On removing the cork a slight explosion and effervescence ensued. Microscopic plants and sporules were observed in it. It had a slightly vinous odour. On applying Trommer's and Moore's tests, I discovered the presence of sugar to a large extent. What! is this a gastric diabetes? can the stomach secrete sugar like the kidney? and is this sugar converted into poisonous oxalic acid? Here was a pretty hypothesis. But in order to test it, I stopped the allowance of sugar in the yellow fever diet and drink, and since then there has been no more effervescence, nor torulæ, nor sugar, in the black vomit. The black

vomit serum always responds to the tests for hydrochloric acid; but as marine salt may always be expected in such a fluid, the results are subject to fallacy in the hands of untrained operators. I therefore entirely distrust any of our chemical researches on this branch of the subject, except those of the most simple and obvious kind, and will not even recount the many experiments that were undertaken with a view to determine the nature of the acid or acids contained in white and black vomit. I, however, forwarded to Dr. Davy a bottle of the fluid, and received from him the following result of his examinations, as contained in a letter from him, dated the 27th of December, 1852, of which the following is an extract:

"The black vomit was not, I think, much changed. When the cork was withdrawn no air escaped; on rest, and many days were necessary, it separated into a pretty clear brownish fluid, and a blackish sediment. The fluid I found of specific gravity 1·049. It became slightly turbid when heated, denoting the presence of a minute quantity of albumen, which was re-dissolved on rest, and probably in consequence of the acid present. The nature of the acid I endeavoured to ascertain. I think I may say it was principally the muriatic, with a trace of sulphuric. I could not satisfy myself of the presence of either the acetic or lactic. Quinine I detected as well as starch, with the former of which no doubt the sulphuric acid had been introduced. Muriate of ammonia I also found in the solution, and in a notable quantity. I could discover no traces of urea, or lithic acid, or of oxalic acid. The sediment—the black matter—was small in quantity. When dried, it weighed only two grains. Under the microscope it exhibited no well-marked or distinctive character. Incinerated, it left a comparatively bulky ash, the greater part of which was not soluble in an acid, and seemed to be chiefly siliceous, no doubt derived from food. The weak acid solution contained a little iron and phosphate of lime, such as the colouring matter of the blood yields when similarly treated."

In corroboration of some of the results of this analysis, I may mention that I have repeatedly examined the serum of black vomit for urea and uric acid, and have uniformly failed in detecting either by the usual chemical modes of procedure. However, as in the case of Brown (Seaman's Hospital, 23rd of October, 1852), on evaporating a drop of the serum in the sunshine on a plate of glass to dryness, not only were crystals of muriate of ammonia noticeable, but also dagger-shaped and crosslet crystals of muriate of soda were found, the form which that salt is said to assume in the presence of urea. In the case of Sullivan (Seaman's Hospital, 3rd of October, 1852), a distinct urinous odour was perceived by all present at the distillation of his black vomit. I had been, for a considerable time, watching for manifestation of the effects of renal obstruction on the blood and excretions. It was on the 1st of October, 1852, that, notwithstanding our repeated failures to obtain urea or uric acid from the black vomit, and the acid character of that fluid, that we thought of questioning it for ammonia. The following is the entry in my notes of the result: "To-day, at the Seaman's Hospital, to the filtered liquid of black vomit (which was highly acid) I added in a test-tube an excess of liquor potassæ. On carefully introducing a test-paper into the vacant space it became blue, and a glass rod with muriatic acid showed white fumes. This was the black vomit in Wood's case. On application of heat to the tube the ammonia was evolved abundantly."

The presence of ammonia in black vomit is universal, that is, it has always been found when looked for, and may be considered one of the tests of black vomit. *White vomit* also contains it in a notable quantity. The specific gravity of black vomit was frequently ascertained, and found to vary from 1·004 to 1·006 at the usual temperature of the air at noon, 86°. Its variations in density were no doubt chiefly occasioned by the accidental admixtures of fluids drunk. Occasionally it was slightly bloody, and then it was coagulable. But in general, the acid of the black vomit seemed adequate to the precipitation of all the albumen. And thus we have had the paradoxical condition of an animal fluid containing hæmatosine without albumen. The sediment of black vomit seemed to consist of coagulated albumen and the débris of blood-cells. In no case in which the black vomit was normal to the eye, was a single perfect corpuscle observed. When pressed through a paper filter the colour is rendered considerably paler. The sediment of black vomit seems more highly acid than the supernatant liquid—it makes a stronger impression on the test-paper. The sediment acts as a ferment on liquids containing sugar. In samples which have been filtered and neutralized to excess with aqua calcis, and put aside, its condition of alkalinity will persist. But in the unfiltered portions, in a few days, the acid re-action will be fully restored. The sediment behaves, under chemical re-agents, similarly to the albumen of the urine. It is dissolved by liquor potassæ, and restored by nitric acid. Several instances have occurred in which black fluids have been ejected from the stomach, and mistaken for black vomit. On the 29th of February, 1852, while passing through the wards of the colonial branch of the hospital, wherein there were several cases of yellow fever, an intelligent nurse brought a basin nearly full of what he considered black vomit, which had been vomited by a Portuguese boy who lay huddled up in bed, seemingly very ill. He had been admitted the previous day for anæmia, and had taken for it the compound steel pill of the hospital. Now although the matter vomited was *blue* black instead of *brown* black, and had not that division into sediment and liquid usually observed, and although the patient's tongue was stained inky, yet a careless and inexperienced observer might possibly have mistaken the ferruginous dose for genuine black vomit. Other instances less palpably fallacious might be adduced of error of observation in this particular, and it is evident that *tests*, independent of mere superficial appearances, are desirable to ascertain the presence of this peculiar and significant product of the stomach—the more so, as traces of it may be present wherein there is no discoloration of the vomited matter, as when the blood corpuscles or hæmatosine has become enveloped in mucus, and thereby kept apart from the action of the acid—cases of which I have seen. The first test should be for its acidity; and, if found, the test is, *pro tanto*, corroborative. But instances, not many, to be sure, but several, will be mentioned in which the fluid ejected from the stomach, and pathologically identical with black vomit, was strongly alkaline. The second test is the solution of the sediment by *liquor potassæ*, which gives the fluid a port wine colour, and brings it out in fluid otherwise pale; when grey mucous flocculi entangling the blood have

hitherto suppressed the true black vomit colour, (as in the case of R. Stopsford, Seaman's Hospital, 13th of February, 1853.) The restoration of the sediment by nitric acid is further corroborative of this test. The third test is the disengagement of ammonia from the fluid by the addition of an excess of liquor potassæ when the black vomit is acid, and by heat alone in the exceptional cases wherein the black vomit is alkaline. This third test may be considered pathognomonic. There is sometimes, in the early stage of acid elimination, a vomit which might be mistaken by the inexperienced eye for black vomit. It has a dark half-floating sediment. This, on examination, will be found to consist of epithelium and mucus, the former stained with bile, the true colour of which comes out under the microscope. In the same stage the vomit also is sometimes a *glairy* acid fluid, with greyish-black tenacious sediment (as with Robinson, Seaman's Hospital, 3rd of January, 1852), which is neither dissolved by liquor potassæ nor shows the port wine tint. The microscope reveals its nature. Like the former, it also consists of epithelium, tinted with bile and closely invested with mucus. The light flocculent matter frequently found floating in genuine black vomit is always mucus, entangling various substances. In one case which I examined, milk globules were found. The patient had drank tea a short time previously. *Normal black vomit* may be described as having a laminar or granular sediment, of a deeper or paler shade of brown, sometimes verging on jet-black, with a clearly-defined supernatant serum of low specific gravity, and without mucosity, partaking of the colour of the sediment, but sometimes nearly limpid when the sediment is black (as if all the colouring matter had subsided). Many deviations from this standard occur from causes already alluded to, such as the presence of ingesta, hæmorrhage, and excessive secretion of mucus. In one case (that of L. Valdon, Seaman's Hospital, 27th of August, 1852), both serum and sediment were bile-tinted. There are, however, two singular varieties worthy of particular remark, though appearing rarely: they may be called the "*caddy black vomit*," although they rarely contain sediment like the alvine evacuations of that name. The first two cases which happened were probably those of Smith and Myhal (Seaman's Hospital, 7th of February, 1852). But at that time the peculiarities were not duly appreciated. The next case was in private practice, in the person of a Mr. Dods, the mate, and subsequently, before his death, the master of the *Grafton* (29th of June, 1852). The next were in Theodore Ternaben (Seaman's Hospital, 4th of September, 1852), and Josea Joachim (Colonial Hospital, 7th of December, 1852), and the last in the steward of the *Livonia*, (in private lodgings, 25th of December, 1852). This vomit does not persist. It appears but once or twice in the individual, and is succeeded by or alternates with normal black vomit. It is of a dirty grey-brown colour, rather homogeneous in appearance, about as thick as mucilage, rather opaque, contains *vibriones*, and is generally strongly alkaline; but may be acid, as in the case of Ternaben. It would seem as if the ammonia in such cases was formed and poured out in excess of the acid, or that the acid was deficient in normal quantity. Josea Joachim's breath was tested, and found highly alkaline. The application of heat to his vomit, without any addition, caused the evolution of copious ammoniacal fumes. In Terna-

ben's case liquor potassæ rendered the vomit transparent, produced a claret colour, and the specimen gave off ammonia, as usual. The microscopic examinations were made within three hours after the vomit had been ejected, quite as early as was the practice in other cases, and yet no vibriones have been observed in the other varieties of black vomit, not even in specimens which have been put aside for several days. In Ternaben's case it is perhaps not correct to call the animalcules *vibriones*. Some of them were globular, about a quarter the size of a blood corpuscle, and some linear, but the latter seemed to be formed by the attachment of four or five of the monads. The movements were very brisk when the light was strong. Were these animalcules the cause of the change in the appearance of the black vomit, or had the excess of ammonia that effect? I incline to the former opinion. For the acid black vomit of Ternaben had the same aspect, and although carbonate of ammonia has been frequently administered internally, no such condition of the black vomit ever resulted therefrom. Moreover, twenty-two hours after the death of Josea Joachim, I had his stomach opened and a sample of the contents removed. The same description of fluid was then seen, but it was *acid*. It refused to yield ammonia till after the addition of *liquor potassæ*. He had taken no acid food or drink of any description before death. The specimen removed had a slight sediment on standing for an hour. It had a strong, unpleasant, somewhat fetid *garlic odour*. The sediment contained only a few shreds of broken epithelium and cell-walls, but *the whole liquid* swarmed with *vibriones*, and their number in this instance undoubtedly communicated to the fluid its dirty greyish-brown colour. The stomach was found coated with the usual tenacious black vomit lining. This condition of the vomit had its counterpart in the urine, as already noticed.

During the former epidemic it was noticed in cases of black vomit, that when it *preceded* the yellow suffusion the prospects of life were improved. The relations of this fact were not then understood. Black vomit is significant of imminent danger, from the circumstance that it is the *dernier ressort* of nature to relieve that contamination of the circulation which has been produced chiefly by impairment of the function of the kidney, and the retention thereby, within the system, of the worn-out nitrogenous elements of the body and their poisonous metamorphoses. Now, if black vomit appear early in the disease, before its march has extended to the great internal viscera, before the bile function has been disturbed or the urine rendered albuminous, it ceases to be the significant symptom which has obtained so much ill-omened celebrity. It is then the sign of a local, instead of a constitutional affection. I have now before me notes of four cases in private practice of what might be termed *benign* black vomit—those of Miss G., Miss S., a Portuguese woman, and a German baker. All these cases terminated in recovery. As yellow fever cases, they were nearly all anomalous. Miss G. had no fever, but strong supra-orbital pains and albuminous urine. Miss S. had one day fever like a paroxysm of intermittent, and the mouthful of black vomit the same day. The Portuguese woman complained only of *malaise*, and on the second day brought up black vomit. The German baker (was three months in the colony: his

comrade, who arrived at the same time, was already dead, from the epidemic,) had fever, but the symptoms were mild, and on the 3rd day he vomited black vomit, *without having had albuminous* urine previously. In such cases the quantity is generally small, and is rarely vomited a second time. Four anomalous cases also occurred in the hospitals during the time included in this report (eighteen months), in which black vomit *preceded* albuminous urine. One was in an anæmic Portuguese, fatal, and presented an extraordinary instance of *truly discoloured* blood after death, —for scarce a trace of even a cell-wall could be found in the port wine-looking sediment. In another (Colin Knoley, Seaman's Hospital, 14th of January, 1853) the black vomit was *succeeded by white vomit.* One of the other two (Reid and Murphy, Seaman's Hospital, 24th of February, 1853) had early black vomit without albuminous urine, seemed to convalesce, but subsequently got albuminous urine and black vomit, of which he died. A post-mortem examination revealed that the first black vomit was probably occasioned by a hæmorrhagic extravasation at the juncture of the œsophagus with the stomach. Among the hundreds of cases of black vomit which I have seen since my attention was directed to the urine-symptoms in yellow fever, those cases just enumerated are all in which a palpable affection of the kidneys was not antecedent to the vomit. But exceptional and anomalous though they be, as Reid's vomit answered to all the tests of genuine black vomit, there is still some mystery about this subject, and perhaps grounds are furnished for the belief that the yellow fever poison acts not only secondarily, in obstructing the liberation of the effete materials of the body, but also directly, in augmenting their quantity. During the first eighteen months of this epidemic there were three cases of chronic disease in which life terminated, to the surprise of all around, with black vomit: these were Dr. B., Mr. B., and Mrs. H. There had been no antecedent fever in either case, nor a single sign of yellow fever that had been recognised. In one of these cases I did not know the condition of the kidneys, and cannot now ascertain, but in the two others I am aware that suppression of urine had occurred for several days before the appearance of the vomit.

CHAPTER VII.

There were many opportunities for becoming acquainted with the condition of the blood during this epidemic. Cupping, the use of the artificial and natural leech, arteriotomy, in a few cases venesection, epistaxis, and other hæmorrhages gave ample opportunity during life for examining its physical, chemical, and microscopic qualities. In no instance could we discover any really abnormal condition of colour, corpuscles, serum, and crassamentum during the first stage, except sometimes the presence of bile. In Mr. Dod's case before mentioned, in consequence of the intense congestion of his face, I opened the temporal artery on the second day of his illness. The blood (three ounces) was florid and coagulated well. The serum alkaline. A little nitric acid dropped into a small portion of it caused instant coagulation, at first opaque white, but after a few minutes a bright yellow, with a *ring* of *purple.* Bile had passed off freely, and until the fifth day, that of his death, decided yellow suffusion could

not be observed. The artery burst open several times before his death, and could be with difficulty restrained, but the blood itself showed no abnormal appearance, except that the bile-test became more decisive up to the time of his death. In every instance in the first stage, the blood retained its normal alkalinity. The changes in the blood therefore were found only in the last stages, and in the post-mortem blood. And yet cases, terminating fatally after normal black vomit and hæmorrhages, as in Flynn (Seaman's Hospital, 14th of December, 1852), G. Ball (Seaman's Hospital, 13th of November, 1852), John Knowles (Seaman's Hospital, 13th of November, 1852), H. Stewart (Seaman's Hospital, 16th of February, 1853), are numerous, in which no unhealthy appearance of blood after death could be noticed, except as to the bile-tinge. The following are samples of the entries in my note-book on this subject:—

"*Blood in Yellow Fever.*—Francis Mitchell, to-day, in Seaman's Hospital, had free epistaxis. The blood was florid and formed a good clot. Under Ross' 1-8th inch object glass, found the discs normal. This was his second day of admission to hospital, 5th of December, 1852. . . . *Blood on second day of Yellow Fever.*—To-day I examined the blood of Mitchel, of the *Lucy* (Seaman's Hospital) The appearance of the blood was healthy, so also under the microscope. The *rouleaux* stood almost perpendicular. The blood was obtained by cupping the neck; 7th of November, 1852. . . . . *Blood on the fourth day of Yellow Fever.*—(*After black vomit.*)—To-day I had a few drops of blood drawn by an artificial leech from the nates of Charles Mitchel. The blood was rich and florid to the eye, and under the microscope was filled with apparently healthy corpuscles. So numerous were they that they much impeded the transmission of light, and for the better observing of them, the thinnest portion of the blood-film on the glass had to be selected. They stood up in erect roulcaus; 9th of November, 1852. . . . . *Blood in Yellow Fever after Death.*—In the case of Charles Mitchel, before referred to, there were two considerably sized fibrinous coagula in the heart. On examining the blood taken from the heart, by the microscope, the corpuscles seemed perfectly healthy and numerous. They arranged themselves here and there in rouleaus. The blood was neutral to the test paper."

The appearance of yellow fibrinous coagula in the heart was frequent after the worst cases, and in fact what may be termed the texture of the blood, often remained good. The following is my note on the case of Michael Flynn:—

"*Blood in Yellow Fever.*—To-day, three hours after death of Michael Flynn, I had an ounce-and-half phial four-fifths filled with blood from his heart. Two hours afterwards I examined the phial, and found the crassamentum so firm as to be unmoved by inverting the phial. On its surface was a bright crimson pellicle, concave, with the limits of the concavity extending about 1-5th of an inch up the side of the phial and covered with serum to the same depth. The serum was slightly alkaline. On forcing a pipette down through the crassamentum, I obtained a particle of blood for the microscope. The corpuscles were found flat and dark in their centre to half their radii, as if from collapse of the centre. Although one or two seemed ruptured, the rest of the discs were perfect in outline. On examining the phial three hours afterwards, at 8 P.M., I found that the clot and serum had still further separated, each occupying about a half of the space of the phial. The serum was a little turbid, and the crassamentum apparently softer. On examining microscopically now by the light of an Argand lamp, transmitted through a 'bull's-eye,' scarcely any of the discs had central darkness—neither were any ruptured cells visible. But about half the number of corpuscles had lost their perfection of outline, and were jagged by nucleoli on their edges, and the number of these seemed to increase during the act of observation. At the side of the thin glass, at which

a little salted water had been applied, the corpuscles arranged themselves in rouleaus; in another specimen, however, they were numerous, without the use of salted water. In this specimen some imperfect or burst cells were seen. The serum when heated coagulated firmly and gave off no ammonia, with or without liquor potassæ. I saw nothing in this blood decidedly abnormal either in colour or physical qualities or minute organism: at any rate, nothing to countenance the idea that the blood was 'dissolved' or even seriously injured by the progress of the disease; and in this aspect of the case—the result of these observations,—I have been considerably disappointed; for the passive (?) hæmorrhages before death led me to expect a serious change in the circulating fluid; 14th December, 1852. . . . . *Death in Yellow Fever.—Blood normal* in colour and consistency, in the case of John Knowles, Seaman's Hospital. I examined his blood to-day, immediately after death. The heart was gorged. The clot was firm, and in due proportion in the test tube when it cooled. The corpuscles were not to be distinguished from those in perfect health; 13th November, 1852. . . . . *Blood of George Ball.*—Two hours after death still warm. A clot of yellow fibrine, about half-an-ounce in weight, found in heart. The serum of a deep yellow bile-colour. The blood clotted firmly. There was an abundance of albumen. The corpuscles normal, except a little bossed or convex in centre, as if the cells were distended; blood slightly alkaline—did not yield ammonia by heat alone, and not much on the addition of liquor potassæ; although comatose before death, his breath was strongly alkaline." Gibney (Seaman's Hospital, 21st of September, 1852) may be cited as another instance of healthy fibrine in fatal yellow fever.

There is, therefore, no doubt of the fact that the blood in the first stage of yellow fever has no appearance of being unhealthy, except as to its occasional intermixture with bile; and also that in many cases which terminate fatally, and with previous black-vomit, the blood is found normal in all its appreciable qualities, except as to the before-mentioned intermixture. But in order to arrive at this conclusion, the blood specimen must be procured direct from the proper containing vessels. If instead of examining the blood of epistaxis in yellow fever we take as our specimen that which has been discharged from the rectum, or by hæmatemesis, when the acid elimination has been scant and the blood little acted on, a very different condition will be found, as was observed in the cases of Jackson (Seaman's Hospital, 21st of April, 1852), Macnamara (Seaman's Hospital, 4th of November, 1852), and Racy (Seaman's Hospital, 7th November, 1852). The following memoranda will serve to describe the condition of the blood in all these cases:—

"*Examination of Blood of Hæmatemesis of Yellow Fever.*—To-day Macnamara, in Seaman's Hospital, vomited about four ounces of mere blood. It was a thin dark fluid, without fibrine—was strongly alkaline—in appearance like port wine. He had had creosote and soda prescribed for irritability of stomach, but none had been given before the blood was ejected. He had passed urine moderately (three ounces) about the same time that he vomited. It was acid, coagulable, with a few tube casts. His breath was alkaline. The blood was not brightened by treating it with nitrate of potash and muriate of soda. On gently heating a specimen in a glass tube, without any addition, it gave out ammonia freely. The heated blood showed scarcely as large a proportion of albumen as the urine had done. Three hours after the blood was vomited, I examined it microscopically. There was not a single perfect corpuscle found, and very little débris. The fluid was nearly colourless under the microscope. There were numerous vibriones of various sizes; some 1-3rd the diameter of a blood corpuscle in breadth, and twice as long as the diameter of a blood corpuscle; others not half this size. They were equally broad throughout their entire length, and their motions were vermicular. There were also other little moving translucent bodies, circular or globular, about a

quarter the size of a blood corpuscle. For the sake of comparison, I at the same time examined some blood of epistaxis which I had brought home from the Seaman's Hospital, and put aside in a phial a week before. When the cork was removed, a slight explosion ensued, and the smell was offensive. It was so thick, however, as to paint the sides of the phial when revolved, and of a deep bright red. When a drop of this was examined under the microscope, no perfect corpuscle was seen, but a large quantity evidently of cell-walls, and the hæmatosine was of a deep tint. There were no animalcules in it. 4th of November, 1852."

Blood passed off by stool, though unmixed with, and of good crassitude and colour to the naked eye, is always found under the microscope with all its corpuscles ruptured. In this intestinal blood, I have never detected vibriones. It seems clear that the alteration observable in the blood from the stomach and intestines, is due in great measure to chemical changes which occur after its extravasation. But though the blood within its proper vessels is often found healthy, through the whole course of the disease up to the last moment of life, it is not always so; and in the last stages is frequently found injured in its obvious constituents of fibrine and cells. As illustrative cases of the deterioration of the fibrine element, reference may be made to those of Morgan and Laman (Seaman's Hospital, 21st of September, 1852), James Walker (Seaman's Hospital, 28th of November, 1852), and Antonio Fernandez (Colonial Hospital, March, 1853). In Morgan's case the fibrine was so diminished in quantity as almost to be lost. In Laman it seemed to have, in a great measure, lost its power of fibrillation. In Walker both fibrine and cells suffered. The right side of the heart was full of dark thin blood, without clots or fibrinous coagula. The colour of the blood was of a dirty brown, and entirely fluid; the corpuscles were all altered and mis-shapen. In all cases, however, the albuminous element seemed, by the rough test of its becoming solid by heat to coagulation point, to be sufficient. A kind of medium deterioration occurred in the case of John Savage (Seaman's Hospital, 19th of November, 1852). My first observation on his case was as follows:—

"*Blood in Yellow Fever.*—To-day John Savage (second day of illness), was cupped on nape of neck. The blood was of a bright vermilion colour, with good clot. The half of the number of corpuscles, however, were rough and jagged, apparently from the adhesion of nucleoli, or the splitting of cells. A few were evidently ruptured and torn. About one-half were normal. Perhaps the heat and spirit vapour in the operation had something to do with these appearances, —14th of November.

The next note is as follows:—

"*Blood after Death in Yellow Fever.*—I had an ounce of blood from Savage's heart about four hours after death. There was a fair proportion of clot, but it was soft, though well separated. The serum looked very thin, and on revolving the blood in the phial, the sides were scarcely stained. But as to the clot, the whole looked like port wine and water. A drop taken from the bottom of the phial, on being examined by the microscope was found to contain corpuscles, but not numerous. A few were injured, but the vast majority were normal. There were none having the appearance before imputed to the heat and spirit-vapour. When the serum was heated, it all set into a firm coagulum almost dry. It was only slightly alkaline, and gave off no ammonia when heated. Although the kidneys, as usual, were much gorged, their function had been but little impaired: thirty ounces of urine having been found in his bladder, though none had been passed for twelve hours before death."

But during life, also, the blood is sometimes found altered. Thus, in Jackson, whose case has been already referred to, after black vomit was established, on the 20th of April, though the blood from epistaxis was florid, and the corpuscles were numerous, they were misshapen, and showed no tendency to form rouleaus. But a few were still normal. Next day, when hæmatemesis succeeded, black vomit and slight epistaxis returned, the corpuscles were still more altered,—they became angular and elongated, with scarcely one normal cell. In the case of John Bridges, admitted to the Seaman's Hospital, 9th of November, 1852, a drop of blood taken by the artificial leech showed the corpuscles spread over the field of view like a pavement. They all seemed flat and jammed against each other, so that there was scarcely any current or movement among them. There was not a single corpuscle of normal appearance; there were no rouleaus, but it was evident that the cells were entire. They speedily became rough by escaped nucleoli. On applying a little salted water to the edge of the glass, currents were immediately induced, and the corpuscles became normal and plump in appearance; but in about a minute they all burst, and the field showed nothing but cell-walls. He had had black vomit before admission. His tongue was denuded of epithelium. He had had no treatment, and described his illness as of only two days' duration. In Peter M'Quin's case (who died early, epileptic, 11th of November, 1852), the corpuscles were flat, indistinct, and irregular in shape, with many nucleoli adhering; but on adding salted water, they bristled with nucleoli like mulberries. Manuel Fernandez was admitted to the Colonial Hospital on the 6th of March, with yellow fever. He had been perfectly *blanched* by previous attacks of intermittent fever. His tongue showed no capillary irritation, his face was pale, and his case at first was erroneously diagnosed, judgment having been biassed by the previous history of the patient. He was treated, therefore, in the beginning, for an intermittent attack. After death there was scarcely any yellowness of the skin. There was no *bloodiness* of the integuments in making the sectio, nor of the tissues or any viscus but the kidneys and stomach. Every other part was anæmic. The *liver*, though recorded as "blood-congested," from its deep purplish-red colour, was not bloody when cut into. The urine found in the bladder was highly coagulable, though that passed during life was not so. The blood was *highly* ammoniacal, though not described emphatically so in the report, and *was totally dissolved.* In those specimens which I took away and examined both by natural and artificial light, I failed to detect a single normal corpuscle. When the blood was examined, no decomposition in the body had taken place (seventeen hours after death); the *rigor mortis* was beginning to yield. The blood was like port wine in colour and consistency. In one specimen under the object glass, two or three almost invisible attenuated corpuscles crossed the field of view, but none of any description in the other specimens, and not even the trace of a cell-wall was to be found. Could this total dissolution of the blood have been possible at any instant before death? Was it the joint effect of the intermittent fever, malaria, and yellow fever poison? Was it the solvent power of ammonia? The healthy condition of the blood in yellow fever seems associated with free action of the kidneys, or copious

black vomit and alkaline exhalations of the breath. And the deterioration of the fibrine has an obvious relation to the amount of *free* ammonia remaining in the circulation. The changes in the *shape* of the corpuseles are probably due to alterations in the density and saline constituents of the serum. The blood of the cadaver in this epidemic, was in the vast majority of cases more or less ammoniacal. In the case of Antonio Fernandez, the water used was *soapy* to the feel till the fibrine was washed out. This soapiness was noticed by Dr. Shier, who called my attention to the fact, before he was aware of our former observations on the ammoniacal alkalinity of the blood in these cases. In a few cases, however, the blood was acid, as in that of Roberts (Seaman's Hospital, 2nd of November, 1852); but it was rarely that ammonia was not extricated in any case by the addition of lime. Bile constantly, ammonia almost constantly, and some undetermined acid occasionally, were the only foreign substances which we were able to detect in the yellow fever blood. Of course, others may have been, and were, likely, present. In order to ascertain the proportions of some of the normal constituents of the blood, I requested Dr. Shier to undertake a chemical examination of some specimens, in the Colonial laboratory. He readily consented, and devoted every week-day from the 21st of March to the 19th of April, to the subject. With the exception of one day, I was present the whole time. The following were the results of the laboratory operations on the post-mortem blood, after following, as far as was practicable, the modes of procedure recommended in Bowman's 'Medical Chemistry,' fourth section, On Blood.

Abstract of Ten Specific Gravity Experiments on Post Mortem Blood of Yellow Fever.

Antonio Fernandez,	1·067826+ at 89°	George Cripsey . .	1·062865 at 88°
Maria de Jesus .	1·056998+ 39°	Francisco Marks . .	1·852576 88°
Edward Richardson	1·0654624 86°	Manuel de Silva . .	1·059498+ 86°
George Sacket (boy)	1·04632+ 86°	Juan Paul	1·059137 87
Rich. Hanson (boy)	1·040008 88°	Robert Lawrence .	1·0616605 85

Proportion in 1000 *Grains.*

Water in Richardson's blood	796·522	Oily fat in ditto	·76
Dry matter in ditto . . .	203·478	Total fat in ditto	1·79+
Inorganic saline matter (ash) in dry blood	50·95	Water extractions (minus ash)	3·321+
Fibrine (minus ash) . . .	1·780	Alcohol extractions (minus ash)	1·526
Water in Juan Paul's blood	787·385+	Water in crassamentum . .	723·633
Dry matter in ditto . . .	212·478+	Dry matter in ditto . . .	276·367
Inorganic saline matter in dry blood	37·12	Fibrine (minus ash) . . .	8·38
Water of serum in Antonio Fernandez's blood . . .	895·877+	Inorganic saline matter in dry crassamentum . . .	31·185
Dry matter in ditto . . .	104·123+	Crystalline fat in dry crassamentum	2·538
Inorganic saline matter in ditto	8·516+	Oily fat in ditto	3·501
Albumen in ditto	72·432	Total fat in dry crassamentum	6·039
Crystalline fat in ditto . .	1·03	Iron in dry crassamentum .	·567

The first important fact which was incidentally observed in conducting the experiments, was the rapidity with which the blood decomposed after

being taken from the vessels of the cadaver. It set rapidly, and within half-an-hour all the healthy physiological changes occurred which were to be expected, and soon after decomposition commences. Frequently no separation at all occurred in the coagulum. The blood was taken from Antonio Fernandez and Maria de Jesus in the afternoon, put into beakers, the ground edges of which were greased, and a plate of glass closely applied over each. These beakers were then put into an ice box till morning. Next day the blood of the female was apparently uncoagulated and somewhat fetid, and in the evening was so offensive, that it had to be thrown away. Immediately on the blood being removed from the body on the previous day it became firm, as well in the beaker as in the specific gravity bottle. Decomposition afterwards, no doubt, caused its fluidity and attenuation. In the case of Antonio Fernandez the clot was large, without buffy coat, with slightly cupped surface, and imperfectly separated from the serum, which was of a dark reddish yellow colour. The odour of the blood was then slightly fetid. The serum was removed by a pipette from two specimens, one to determine the proportion of *albumen* and the other the proportion of *water* in the serum. To verify former observations, 31·605 grains were taken for the application of the bile test. But the serum was not perfectly clear, and our inability to obtain the requisite quantity necessarily caused a deviation from the plan prescribed by Bowman for the analysis of coagulated blood. The following note occurs in our journal of the 9th of April:—

"It may be necessary to record that, in estimating the *albumen*, *salts*, and *extractions* of Antonio Fernandez' blood, we could not follow the plan of Bowman in his section on the quantitative analysis of coagulated blood, from the smallness of the quantity of serum obtainable. The albumen was therefore obtained by the method recommended by the same author for obtaining it in diseased blood. The washings are being treated with ether for the fat. But as hydrochloric acid was added to neutralize the alkalinity of the serum, of course no true estimate could be made of the *salts* from that sample. The salts of the serum were therefore estimated from the dry matter obtained in estimating the *water* of the serum. In addition to an estimate of the fat of the serum, we have now under treatment a sample of *dry blood* of Fernandez, to ascertain its proportion of *fat*. We shall proceed afterwards to estimate also the saline constituents of the clot. In reference to the estimate of the *water* in the clot of Fernandez, it may be remarked, that after being weighed it was dried during several days on a chloride of calcium bath, an uniform density of the fluid being maintained by a tube returning the condensed vapour. While drying, the clot was carefully and repeatedly broken up into small fragments to facilitate the escape of vapour, and the drying continued till it ceased to lose weight. It was not minutely pulverized, however, and again dried, as it probably should have been before being finally weighed. No instruction to that effect being given by Bowman, and having ourselves made no analyses of healthy blood whereby a standard might be obtained, we deemed it prudent in this instance not to exceed our instructions, lest comparative results might be affected by a want of parallelism in the methods of procedure."

The present mode of defibrinating the blood is coarse and unsatisfactory, and some new or more chemical method is desirable. While, however, the present methods are pursued, and may be continued for comparative results, the degree of fineness of the muslin bag in which the blood is washed should be stated. In that employed by us in defibrinating the

blood of Fernandez, the tissue contained two hundred threads in the superficial square inch. Richardson's blood was attempted to be defibrinated by agitation with pieces of lead; but finding this mode inoperative, and that very little fibrine had attached itself to the lead, we completed the operation by subjecting the blood to a gentle stream of pure rain water in a *calico* bag, which, when wet, was almost air-tight. I subjected the washings in both instances to microscopic examination, after they had remained about sixteen hours to settle. They were then fetid. In Fernandez' I detected numerous fibrillæ and granules among the wreck of the blood cells, and numerous oval and bottle-shaped polygastric animalcules, which moved with great velocity, and were each about four times the size of a blood corpuscle. There were also bodies in the sediment like quartz-shaped uric acid crystals, but on treating them with nitric acid and ammonia, they did not prove to be such. Some of these bodies seemed black to the naked eye, and bluish black by transmitted light, but by reflected light they showed the same quartz-like lustre and appearance. These, likely, had been stained by the dye of a black or dark-blue string, with which the mouth of the muslin bag had been tied. Richardson's blood had coagulated like currant jelly, and the serum had never separated. It was so ammoniacal, that fumes were detected by the muriatic acid rod at the temperature of the atmosphere, without being heated. The washings of his blood yielded considerable sediment. This sediment had a yellowish, curdy, flaky appearance to the naked eye, exactly like the "curdy" urinary sediment which I had observed, and is described before. The sediment showed no fibrillæ, but had a granular appearance, as if the fibrillæ had been disintegrated, or their granular structure disconnected, which may be the mode of the destruction of fibrine by yellow fever. If so, the amount of fibrine estimated in yellow fever blood by the usual methods must be considered as not the whole fibrinous matter contained in the blood, but only the whole which has escaped disintegration, or which retains its power of fibrillating, and thereby capable of being collected by the present methods. There were some singularities in the case of Antonio Fernandez. He had suffered from an attack of pleuritis extending over about three inches of the left side, coming on during the progress of the yellow fever, and occasioned probably by the situation of his bed, which was in nearly a thorough draught. He also had the scarlatinoid rash more markedly and more extensively than I ever saw before, and it was accompanied by a turgescence of the skin, which subsided on the advent of black vomit. As the black vomit was not very copious, and lasted only one day, and although he lost a considerable quantity of blood by urine, as he had suffered from an inflammatory complication, I naturally expected to find his blood rich in fibrine, and but little deficient in its saline constituents. It may be here noticed, that in obtaining the specimens for examination from the large vessels of the chest, care was taken that no fluid from the pleura, mediastinum, or pericardium, should be mixed with the blood. Some effusion had taken place as a consequence of pleuritis in the case of Fernandez. In washing out the fat from the albumen by boiling ether, the filters on the second day refused to act, probably from an obstruction of their pores, and steam-washing was had recourse to during two suc-

cessive days, until no trace of organic or saline matter passed through, the criterion of which was the stainless evaporation of a drop of the filtered fluid on a piece of glass heated over the spirit lamp. It may be remarked that this criterion would not be applicable to filter-washing with *commercial* ether, which leaves a stain of itself, from probably an impure spirit (containing fusil oil) being frequently used in its manufacture. Without attention to this fact, the ether filtration may go on to an indefinite period—for the stain on the glass will be well marked, and will *volatilize before being charred*, leading to the inference that the fat is not yet completely separated from the albumen, which may or may not be correct. The alcohol used for the *extractions* was of the strength of 45° of Baumé, and four washings were applied.

On the 13th of April, an estimate of the *iron* in the inorganic matter (ash) of the *dry blood* of Fernandez, which had been incinerated the previous day, was commenced. As this analysis was independent of the instructions contained in Bowman's 'Medical Chemistry,' it will be right to describe the method pursued:—To the ash was added an excess of muriatic acid, and then it was digested in a bath till solution was effected, and a few drops of nitric acid were added to peroxidise the iron. It was afterwards filtered to free it from some particles of impurity, and treated with ammonia for precipitation, and allowed to remain till next morning. The ammoniacal precipitate of iron was then steam-washed till only a trace of stain could be detected on evaporating a drop of the filtered liquid on a piece of glass. The filter (No. 5) and its contents were then dried in another filter paper, so as to be removed into an agate mortar, when it was mixed with an excess of *pure* carbonate of soda. The filter paper was then ignited and placed in a counterpoised platina crucible, and further incinerated over a spirit-lamp, and then mixed with an excess of *pure* carbonate of soda, and added to the other portion so treated. The whole was then fused, and after fusion lixiviated with distilled water and left till next day. On the following day, after maceration, filtration, and steam-washing on filter paper of known weight, the precipitate and paper were dried and weighed in a counterpoised tube. The net weight of the iron was found to be ·23 of a grain. The great care in removing the phosphates may show a weight of iron comparatively light. It may be remarked, that throughout the analysis of Fernandez' blood, the phosphates caused much trouble and delay. A crucible could never be used a second time after simple washing, or the use of an acid. Fusion by microcosmic salt, or other flux, was required after each incineration. Notwithstanding this strong crusting of the crucibles, the ash of the several incinerations was very deliquescent, no doubt from the presence of potash. After deliquescence spangles were seen over the surface. On examining one sample of residuary ash on the last day of operations with a one-inch object-glass, I found that these spangles were long flat prisms, associated with amorphous opaque crystalline matter, tinted brown, probably from the presence of iron (it was the sample from the albumen washing). The whole of the prisms rapidly disappeared on the addition of nitric acid, and a great part of the amorphous matter. An accident happened to the specimen, when ammonia was added and the glass put in the sunshine. So I cannot say that the crystals were restored.

It is probable, however, that the prisms were triple phosphates, and the amorphous matter phosphate of lime. The estimate of *water* in Richardson's blood appears high. This may be in part owing to the extreme care with which the drying process was conducted. A water bath was used in the first instance, and the mass was then pulverized, and the drying finished off with a heat of from 230° to 240° in a chloride of calcium bath.

In extended investigation on the specific gravity of yellow fever blood, a law of age and sex might be evolved. It may be noticed in the instances already given, that the lowest gravities are in the boy and woman. Other circumstances being equal, the copious ejection of black-vomit should increase the density of the blood by diminishing its proportion of water, and this view would seem to be borne out by the converse fact that Francesco Mark, who died without black vomit, has blood of the lowest density of all the adult males; and Juan Paul, who had but little black vomit before death, is the next lowest in the same category. In Francesco Mark's case, the specific gravity bottle was filled seven and a half hours after death, and the blood set firmly in it. Robert Laurence was fifty years of age; the specific gravity bottle was filled twelve hours after death, and was weighed two hours afterwards. The blood had then set firmly without separation of serum. He had had copious black vomit before death. In George Crispy's case, the bottle was filled eight hours after death, and the blood set well in it. A quantity was also placed in a small beaker, and twenty-four hours afterwards a soft coagulum filled the whole space occupied by the blood, without separation at the sides. But there was on the upper surface about half-an-ounce of very dark serum, with floating pellicles. This serum may have constituted about $\frac{1}{10}$th of the whole. The blood smelled decidedly *urinous*. On examining it with Pritchard's $\frac{1}{4}$th or $\frac{1}{3}$rd inch object glass (for the power is not stamped on the case) the pellicle seemed to consist of granules, such as are seen in the curdy sediment of albuminous urine. There also appeared what seemed to be oil globules, and other bodies which were not round, but somewhat prismatic in shape and luminous in centre, such as are delineated in drawings of hippuric acid. As regards P. Burke, on the 12th of April, 1853, I tried to take the specific gravity of his blood two hours after death, but it coagulated so rapidly that it could not be got into the bottle in a homogeneous state, and there was an excess of serum introduced. It was weighed, notwithstanding, and its specific gravity in the condition stated, was 1·04508, at a temperature of 87°. In the bottle the serum separated clearly and in large quantity, and was as usual alkaline. He had had copious black vomit before death. Some of his blood was also set up in a beaker, and after twenty-four hours inspected. It then had the clot and serum separate; but the clot was very weak and the serum of a bloody orange colour and turbid, from bile and the hæmatosine of ruptured cells. No fetor was observable. On the 9th of April I wished to take the specific gravity of the blood of Charles Bush, who died in the Seaman's Hospital on the previous day, in order to compare it with that of Francesco Mark, the former having had copious black vomit before death. But he had already been dead twenty-four hours; and very soon after the blood was put into the bottle it began to bubble with gas, and actually frothed soon after. When weighed while

the bubbles of gas were rising, its specific gravity was still 1·050, and in this state of decomposition it was very fluid, and readily flowed out of the bottle when inverted.

Numerous quantitative analyses of the blood of yellow fever would obviously be of the greatest importance to the elucidation of the pathology of the disease. But such an undertaking is impracticable; though a working chemist, associated with an intelligent physician, might well occupy five or six years in such pursuits to the great advantage of the whole tropical world. In the meantime much valuable information might be added by the zealous practitioner to the general stock, by multiplying accurate observations of the blood's density, and its alkalimetry according to Griffin's method; these, with a table of some of the most important symptoms, and the age and sex, &c., of each patient, attached, would yield excellent results.

CHAPTER VIII.

Immediately after becoming acquainted with the discovery of Frereichs, regarding the conversion of urea, its applicability to our yellow fever investigation was at once apparent, and I forthwith proceeded to the Seaman's Hospital, with the view of ascertaining if the *breath* of the patients in the advanced stages would give any signs of the presence of ammonia. The attempt failed on that occasion to afford any indications; and a note was not even made of the experiment as to the date or result. The impression for the time was fixed, that the urea necessarily in the blood in cases with suppression of the function of the kidney remained unchanged. Up to this period the alkalinity of the blood, which had been frequently observed, was supposed to be that of its normal condition, and our object of research was chiefly the recognition of its departure from this state—its acidity. The examination of the blood, and the detection of ammonia in it, in the case of Lannan, renewed our attention to Frereichs' theory, and we argued that the carbonate of ammonia might be formed, but concealed by a nascent combination with some acid. The next experiments were entirely successful.

The following is my note of it:—

"*Carbonate of Ammonia detected by me to-day in the breath of Stress and Whittaker, both of the Seaman's Hospital; yellow fever cases.*—I found that Stress had passed about one ounce of urine to-day, which was rather mucous, with a sediment of epithelium tube casts, and several *crystals of triple phosphates*. It was highly coagulable. After allowing the coagulum to subside and separate, I took a watch-glass, and reduced the clear urine slowly by the heat of a spirit-lamp to one-third its bulk. I then added an equal quantity of nitric acid, and placed the watch-glass on a piece of ice. The experiment was carefully repeated thrice. A mere trace of nitrate of urea was obtained. At first it was supposed to be entirely absent, and it is so entered in the case-book; but on adding alcohol, and then evaporating a drop on a piece of glass, traces of the nitrate of urea were observed by the microscope. The case of Stress at the time of observation was that of incipient black vomit. He was distressed, breathing quickly, and somewhat nasal. I moistened a neutral test-paper (Griffin's), and held it to his breath for about half a minute, when it became blue. I then held a glass rod, dipped in muriatic acid close to his mouth, when white fumes were visible. Whittaker's case was more advanced. He had intense black vomit. Eye yellow; respiration nasal; inclination to restless torpor. No urine to examine. The instant he breathed on the

wet test-paper, it became blue, and muriatic acid showed white fumes. I prescribed lemonade *ad libitum*, with a little sugar, and immersion in an acetic acid tepid bath, of the strength of about one-sixteenth of vinegar to the whole bath. 26th Sept., 1852."

After this, the investigation was quickly followed up, and it became apparent that the urea of the suppressed urine is eliminated from the system as a volatile salt by its metamorphosis into a carbonate of ammonia, which, as such, is frequently found in the breath, occasionally in the black vomit and hæmatemesis, and almost always in the stool, twice in the urine (Ellwood and Macey), always in normal black vomit in combination with an acid; and, indeed, apparently pervading all the tissues of the body.

We have made many attempts to detect urea and uric acid in the circulation and in the serum of the ventricle of the brain, but uniformly without success. This certainly may have arisen from the incompetency of the operators, and subsequent manipulation may accomplish it. Still, it seems probable that the mode by which an attempt is made by nature to unload the system of the urea when its natural channel is obstructed in yellow fever, is, failing a restoration of the function of the kidneys, the conversion of this substance into ammonia, which is eliminated in the manner before described. The uremic condition of the blood seems a fugitive affection. In the case of Flynn, before referred to, it seemed to have passed off before death. The blood was only slightly alkaline, although the breath, when examined, was highly ammoniacal. But by the breath, black vomit, and a partial restoration of the function of the kidney (he passed on the day of his death eight ounces of urine, specific gravity 1·033, at 80°), the circulation had been relieved. Such was the case also in George Balls. His blood was tested for ammonia, and found free of it after death, although during life the breath had been highly ammoniacal. Josea Joachim, before referred to, on account of being a subject wherein the peculiar black vomit appeared, and whose breath and vomit showed a saturation of the system with ammonia during life, had large fibrinous coagulæ in his heart after death, and no *free* ammonia in his blood. The blood was acid, and ammoniacal vapours were produced only after the addition of *liquor potassæ*. In this case the system seemed relieved of its urea by a tertiary combination—the formation of a neutral salt by the ammonia with an acid (phosphoric?). Sometimes, where you have suppression of urine and symptoms of uremic poisoning, you may find little or no alkalinity of breath, as in the cases of Clarke (Seaman's Hospital, 19th of October, 1852) and Walker (Seaman's Hospital, 28th of November, 1852). In some of these instances I can suggest no explanation but that urea of itself is adequate to all the phenomena, as originally supposed, or that sometimes the feebleness and *shallowness* of the expiratory movement, when the lungs are undergoing engorgement, and the diffusion of the exhaled air through both mouth and nostrils, may prevent the test-paper or glass rod from being affected. I have been in the habit, from the latter consideration, of always closing both nostrils with the finger and thumb, and permitting the patient to breathe through the mouth only while the test is being applied. The degree of alkalinity may be roughly estimated in these experiments by the number of expirations

required to strike a distinct blue on the moistened end of the test-paper. Uræmic symptoms are most severely and distinctly brought out when the urine is suppressed after black vomit, has commenced copiously and afterwards ceased, and the mucous diarrhœa also ceased, as in the cases of Mr. Glynn (at Mr. S.'s), Mr. B. J., the cook of the *Susan* (Seaman's Hospital, 5th of February, 1852) and a mate about the same time. Then, instead of the placidity of mind and freedom from suffering for which the fatal termination of yellow fever is often so remarkable, the whole train of manifestation is changed. The pulse, instead of its usual loss of power and threadiness, revives or remains full and strong, the pupils become contracted, the eye sometimes again assumes a glistening appearance, and delirium, convulsions, fearful shrieks, and stertorous breathing may close the dreadful scene. Of course this condition varies in degree, sometimes amounting only to uremic intoxication, in ratio with the extent of the locked up secretions. In the protracted case of the mate of the *Sabraon* (private practice; black vomit on the eleventh day, and death on the thirteenth) the uremic symptoms appeared on two occasions. At first they occurred before the black vomit. They were marked by apathy and despondence, to which succeeded low muttering delirium and *subsultus tendinum*. This condition lasted two days, after which black vomit ensued in immense quantities, and *forthwith the intellect became perfectly clear*, and the subsultus much diminished. But an incessant hiccup supervened. The vomit changed into a port wine-like fluid, at first a little acid, but in a few hours alkaline, and it emitted ammonia on the application of heat without *liquor potassæ*. Under the microscope not a single entire corpuscle was to be seen, but much *débris* of blood-cells. The urine had been tolerably free throughout this protracted case. On the day before his death he passed twelve ounces. It was highly acid—rather turbid, with a thin layer of blood corpuscles, and with a very few amorphous and not large masses of the material of tube casts. Its specific gravity at 86° was 1·018. It was highly albuminous. After being heated to evaporate the albumen, it was then carefully tried for urea, and the evaporation conducted very gently. It formed no crystals of the nitrate of urea. But a trace was discovered in the mucous-looking ring, at the edge of the watch glass, by alcohol, under the microscope. It was tried thrice with the same result. Twenty-four hours before death the urine became suppressed; vomiting also ceased. He then became exceedingly restless; much jactitation, intolerable sense of internal heat complained of, beginning in the throat and epigastrium, and subsequently extending to the feet and hands, while the surface is actually cool. Refuses everything. Tongue, that was yesterday clean, now dryish and incrusted with blood. Delirium and coma closed the fatal scene at midnight, 23rd of January, 1853. This modification of the uræmic symptoms in the first instance may have arisen from a moral cause. This patient was doing well, and apparently convalescent, after having had black vomit *incipiens* on the fourth or fifth day. But the master of the vessel visited him, and rudely reproached him for the expense he put the vessel to, by his lying up in private lodgings, instead of having gone to the hospital. He visibly took these taunts to heart, and the unfavourable and fatal symptoms commenced directly.

In general, the yellow fever cadaver remains in a suitable condition for dissection as long as that of any other disease. But when the urea has not got vent during life, and the putrescent elements are retained, decomposition is rapid. A case in point was that of Tomlinson (Seaman's Hospital, 9th of October, 1852). The following is my note:

"*Singularly Rapid Decomposition after Death in the case of Tomlinson.*—Yesterday at noon he had no appearance of illness; skin was cool, pulse little excited, although in the third stage of yellow fever. There was, however, an almost total suppression of urine, and his breath was highly alkaline. He died yesterday evening, and this morning the corpse had the appearance of having weltered in the sun many days. It was black, enormously distended, and covered with large vesications. It was in an unapproachable state for dissection. He had had no well-defined black vomit. There had been some white vomit, and about four ounces of imperfectly-developed black vomit altogether; and only one scanty sanious stool of about three ounces. I examined three ounces of urine, which had passed yesterday at two P.M. The coagulum, when heated, did not readily subside, and amounted to about one-fifth of the whole. When tried for urea, no crystals formed. A slight haze, however, formed on the surface, which, when treated with alcohol, showed traces of the nitrate under the microscope. The urine was strongly acid;—9th October, 1852."

In some moribund cases I have observed strong alkalinity of breath, while acid beads of perspiration stood on the face and forehead, which when evaporated and examined microscopically showed, after evaporation to dryness, dagger-shaped and cross-slit crystals, as in the case of Cook (Seaman's Hospital, 9th of December, 1852). But uræmic poisoning is not the only mode of death (see cases, Patterson, Seaman's Hospital, 9th of July, 1853, and Antonio Gonsalvo, Colonial Hospital, same date); nor are the cerebral symptoms always due to that cause. Hyperæmia is capable of inducing similar symptoms, as in the case of Laird (Seaman's Hospital, 16th of October, 1852), in which, indeed, both classes of symptoms were present, but the latter antecedent as to time.

Respiration in the last stage of yellow fever is sometimes very laborious, and frequently at each inspiration the nostrils collapse and shut, and if the half-comatose patient keeps the mouth shut (as in the case of Juan Martinez, Colonial Hospital, 13th of June, 1852), asphyxia may ensue therefrom. It is obviously difficult in many of these cases to refer the symptom to its true cause—to distinguish the effects of a poisonous circulation on the brain generally from those of direct pressure on the medulla oblongata. There is a description of the respiratory act, named, I believe, by Dr. Graves, "cerebral respiration." This epithet frequently occurs in our case books. In many of the cases to which it is applied, the intelligence is not much, if at all, impaired; and the name of suspiration might perhaps judiciously be substituted for that of cerebral respiration. It is a hurried sighing respiration, in which the nostrils also take part; it is frequently accompanied by restlessness and jactitation. In the generality of cases, this state is really independent of all nervous influence, as far as any affection of the body can be, and is the direct effect of congestion of the lungs, threatening impending pulmonary apoplexy.

(*To be continued.*)

CHAPTER IX.

In general, after convalescence from yellow fever, the recovery to perfect health is rapid and thorough. I have notes, however, of five seamen who before discharge from hospital suffered from paroxysms of intermittent fever, in the months of July and August, 1852. In the Colonial branch, at various times, parties discharged cured of yellow fever have returned within from ten to fifty days, suffering from the endemic intermittent. These were chiefly Portuguese immigrants, who are very prone to this disease at all seasons, and during non-epidemic periods. These sequelæ, however, were more common in the months of July and August than in other months. I have a note of only one case in private practice (that of Mrs. M.), where intermittent fever appeared clearly as a sequel of yellow fever. Bloody furuncles, as before noticed, are so close on the primary affection, and so obviously one of its morbid processes, and so frequently co-existent with black vomit (in the case of Mr. L. M., a white native, who died on the third day of illness with black vomit, a malignant-looking furuncle appeared on the upper lip), that it is doubtful if it should be rated as a sequela only. But they frequently appear during apparent convalescence, and of course retard it, and become associated with abscess and ulcers, which readily heal. The parotid gland suppurated in four cases of Portuguese, as the result of a bloody furuncle in each; and in one sailor, Devine (Seaman's Hospital, 17th of November, 1852), there was one instance of a bubo over Poupart's ligament as a sequela. In convalescence, also, small boils frequently appear over the face and other parts of the body, but it is difficult to ascertain whether these should be referred to the previous disease or the irritation which follows the application of vesicatories, and is observed so often as their effect when applied in other affections. Gangrene of the prepuce occurred in the case of Ernest Home (Seaman's Hospital), who was suffering from gonorrhœa before and during the attack of yellow fever. Anasarca of face, hands, and feet, without desquamation of cuticle, while the skin was still yellow, appeared in the case of Grammage (Seaman's Hospital, 2nd of October, 1852). In the case of Mr. Mackinnon, of the *Jane Brown*, urticaria came on while the skin was still very yellow, accompanied with abdominal pains. Then a recession of the rash took place, and dementia (preceded by oscillatory movements of the eyes), quadruple vision, and death. Oozing from the gums was frequent in convalescence from the "smouldering" forms of the disease. Retention of urine occurred in the case of Anderson (the Swede). One seaman was readmitted to hospital on account of debility, after his attack. Where the attack had been severe and profound, wasting of the body was sometimes found to have taken place, as (markedly) in the case of the master of the brig *Speculation*, who had had black vomit before recovery; and Peter Daly, Major, and Anderson, before referred to. When venesection had been used in treatment (as in the case of Havish, Seaman's Hospital, 16th of January, 1853), convalescence was much protracted. Bright's disease was a sequel in the instance already mentioned. The patient, Manuel D'Alvia was admitted to the Colonial Hospital on the 5th of April, 1853,

with a violent attaek of the epidemic, from whieh he reeovered. He was diseharged on the 16th of July, at his own urgent reqnest, with his urine still albuminous. He had been cupped for the sequela over the kidneys: had issues then applied: had taken a long course of gallic acid, but without benefit. His ailment somewhat emaciated and anæmiated him, and gave to his countenance an expression of gravity ; yet there was no œdema or dropsy, and his appetite was tolerably good. As he felt well, he could not understand why he should remain in hospital. The most singular sequela of yellow fever, if it be really one, was that in the ease of Miss G., a subject of one of the anomalous cases of black vomit before referred to. I saw her in consultation about five months subsequent to the period when I had seen her in the primary affection. She suffered from a compound of anæsthesia, pain, and atrophy of the left hand and fore-arm. It began about two months before with numbness, and afterwards with some peculiar occasional pains; but no physical alteration was noticed, and she was supposed to be fanciful. Since then, the symptoms have much increased, with both numbness and tenderness on friction, chiefly along the course of the ulnar nerve. The fore-arm is much wasted, but the startling atrophy is in the fingers. Motion is perfect; there is no breach of surface; but the fingers have an attenuated ivory appearance. She has not been using her left arm, or but rarely, for some time, even before she began to complain. But the wasting is not muscular only. It seems as if all the tissues, and even bones, had wasted symmetrically. She had never been quite well since the black vomit, and has had several attacks of intermittent since then, and since the commencement of the present affection. Chalybeates and galvanism were recommended, and change of climate, which being adopted, the case has been lost sight of, and the result unknown. Two cases of abscess of the liver followed as sequela of yellow fever; one in a Portuguese man, Josia Joachim, admitted to Colonial Hospital, 24th of February, and discharged 28th of May, 1852. In this case the liver was twice opened, and an enormous discharge of purulent matter let out. The other case was in a negro girl, Lucy, a native of Barbadoes, once punctured, and cured. Inflammation, acute pain, and swelling of the joints occur sometimes in convalescence, when the action of the poison has been profound; but though often threatening suppuration, these painful swellings have always terminated by resolution. One of the most common sequelæ is jaundice, and this greatly retards the period of perfect recovery. Most of the cases that remain long in hospital after convalescence, and of which period no report is made in the case books, have been detained by this affection. It is a true sequela, and not to be confounded with the lemon tinge and orange eye which are present in the advanced stages of the disease. As has already been noticed, in this sequela the eye is smooth and unvascular, and the skin yellow or tawny, the urine is copious, and loaded with bile, and the fæces are formed, grey, and abilious. The one state seems the result of *excitement* of the liver, and the other of *obstruction* of the bile-ducts. One of the most uncommon ailments of females, colica pictonum, attacked Mrs. B. in early convalescence from an attack of the prevailing disease, which had proceeded to the stage of acid elimination, on exposure to the atmosphere of a lately painted room.

Relapses were of frequent occurrence, occasioned most likely, in great measure, by a return of the patient to the focus of infection after discharge from the hospital. These relapses were almost exclusively among the aborted cases. They frequently recurred, and were aborted several times. The primary attack was generally without albuminosity of urine, and frequently the relapse also, as in the captain of the *Undine* (private practice), and Thomas Wright (Seaman's Hospital, 17th of June, 1852), &c. &c. But in the relapse there was often an accumulated power in the disease, and albuminous urine was expected, even if the disease were again aborted, during convalescence. Relapses, however, occur in which, and in the primary attack, the urine was albuminous, as with R. Fuyakerly (Seaman's Hospital, 25th of July, 1852), &c., in whom the relapse was easiest of abortion; and in George Macey (Seaman's Hospital, 12th of February, 1853), and Peter Francis (Seaman's Hospital, 5th of March, 1853), in whom the relapse was fatal. These, however, were comparatively rare, and we have had only two relapses after the disease had run on to black vomit—viz., those of Anderson (Seaman's Hospital, 16th of February, 1853), and Adam Smith (Seaman's Hospital, 31st of December, 1852), both of which were readily aborted, although the last-mentioned relapse proceeded to albuminous urine. Duncan Livingston (Seaman's Hospital, 3rd of August, 1852) sustained a relapse or second attack. His first was on the previous 12th of July; and though on that occasion his urine was far from albuminous, the eye was tinged. Daniel Clarke (Seaman's Hospital, 29th of June, 1852), was then in hospital for a relapse, and stated that he was very ill ten or eleven years ago, in Demerara, with yellow fever. On the 23rd of August, 1852, the steward of the *Maria* was admitted to hospital with an attack of the prevailing epidemic, which was aborted by two doses. He stated, that four years ago he was very ill at Vera Cruz with yellow fever, and suffered afterwards at the same place with intermittent fever. Relapses were more numerous than appear in the case books, when they occurred in hospital. Such were promptly and extemporaneously prescribed for; and if they were aborted by the first dose, as they frequently were, no report was made of them. The tendency to relapse or second attack was generally within the first month after the primary attack. Master J. B., and Mr. M. C. (of the house of Irvine and Sons) had each a second attack exactly one year after the first, both recovering. The primary attack in the former having been severe, and the second mild, exactly the reverse of what happened with the last-named patient. A case of yellow fever, alternating with intermittent, and ending fatally, occurred in the Colonial Hospital. Manuel de Frytas, only three months in the colony, had several attacks of intermittent, one of which was on the 27th of October, 1852, but on the 10th of November following he was admitted for an attack of yellow fever, which proceeded on to albuminous urine and scrotal excoriations. He was discharged on the 28th of November, cured. On the 7th of December he was re-admitted for intermittent fever, and cured; and on the 4th of January following he was again admitted with yellow fever, urine highly albuminous on that day, and he died suddenly on the 6th of January. There was no post-mortem examination. Relapses, then, were frequent after aborted attacks, but very rare after the disease ran to its

second stage, whether it stopped with the first stage of acid elimination or proceeded to black vomit.

When the epidemic has terminated, and the harvest of facts are gathered to their granary, then, by the application of the *numerical method* to this and other branches of the subject, the vague terms "frequent," and "seldom," may be dropped, and the ratio of frequency of the several symptoms can be stated with precision. The present estimates are rather qualitative than quantitative.

CHAPTER X.

The mode of death in uncomplicated yellow fever has four distinct varieties, and these are sometimes blended—viz., syncope, uræmia, apoplexy, and asphyxia. When the black vomit is plentiful or the urine free, the intelligence remains clear and unclouded; but the skin becomes cold and damp; the pulse small, and, finally, extinct at the wrist, and the patient dies of gradual exhaustion and syncope. Lamont (Seaman's Hospital, 5th of December, 1852) died apparently from rapid collapse, following excessive discharges of black vomit. The description of the mode of death by uræmia has already been in great measure anticipated in the foregoing chapters. If before death the urine be suppressed, and the black vomit is not copious, or has ceased, the circulation becomes contaminated; and when this condition operates on the brain in its mildest form, the effect is not unlike alcoholic inebriation; as in the case of the master of the *Hindu*, who, on the night of his death, sat up in bed, drank beverages, and joked with the ship-masters around him; and the carpenter of the *Eleanor*, who, within a few hours of his death, and while pulseless, I found, on my visit, sitting up in his chair, and regaling himself with his tobacco-pipe. If all the excretions and secretions be locked up, as occasionally happens (the master of the *Honor*, for instance), the symptoms of uræmic poisoning become violent, the sensorium painfully affected, irritability of temper, screams and wild ravings, coma and convulsions, ensue. Death from syncope does not arise from excessive discharges of black vomit alone. It is often the result of hæmorrhage, as in the case of the uncontrollable epistaxis in W. Smith (Seaman's Hospital, 25th of March, 1852); or bleeding from the mouth and gums, as in the case of Ferguson (Seaman's Hospital, 2nd of November, 1852). Frequently these two causes—i.e., black vomit and hæmorrhage—combine in inducing this mode of death, as in the case of Mrs. W. The following extract note, written on the day of her death, illustrates this point:

"Before black vomit appeared, the catamenia came on prematurely, the bowels became spontaneously relaxed; and last night there was much flatulent purging of blood, and a considerable hæmorrhage from vagina. After a cessation of twenty hours, black vomit again returned to-day. After total suppression for twenty-four hours, four ounces of alkaline urine was drawn off by catheter. On my visit at daylight this morning, she was quiet, and apparently suffering no pain, and rather apathetic. The marked change which I found in her case, was a deterioration of the pulse in volume. The nervous symptoms of the preceding day, which threatened inebriation or convulsions, had disappeared. The pulse became gradually weaker, until about eleven A.M., when it could not be felt. She was aware of her hopeless condition, and tranquilly disposed of her trinkets to her friends and

relations. As she approached her end, the breathing became quicker and shorter, until it ceased in a few little gasps at long intervals. About half an hour before she died, she apparently lost her vision, then her hearing, and sensation, first of the mouth and nose, and then of the arm, in quick succession, and in the order stated. It was an appalling scene, to see her lying silently on her back, and trying to *rub back* vision and hearing and feeling, with her hands. She spoke not a word during the time; but it was evident that the senses were all being blotted out one by one while consciousness yet remained. Before death, at two P.M., one or two slight convulsive jerks of the shoulders were the last respiratory efforts.—17th of February, 1853."

After death in this case, a large quantity of black vomit escaped from the mouth in turning the body. The mode of death by apoplexy, caused by congestion, and effusion and extravasation of blood on the brain, is instanced in the Seaman's Hospital cases of R. Williams (3rd of March, 1852), Peter M'Guire (13th of November, 1852), and Peter Thomas (17th of December, 1852). The following Seaman's Hospital cases furnish instances of death by asphyxia:—Moses Dillon (25th of July, 1852), laryngeal suffocation; the Portuguese sailor (31st of August, 1852), and Milligan (14th of December, 1852), from pulmonary apoplexy. Cases sometimes terminate suddenly, as if by *explosion*. Thus, Peter Scott (Seaman's Hospital) was doing well. On the 1st of November, 1852, at noon, he suddenly became ill, vomited black vomit, and died within a few hours, with alkaline breath. Patterson, who died on the 18th of February, 1853, had his stomach perfectly quiet till within a few hours of his death, when he suddenly disengaged immense quantities of black vomit. His urine had been tolerably free up to a short time previously. Alexander Stewart, who died on the 14th of February, 1853, in Seaman's Hospital, is another instance. He became suddenly ill at four P.M. on his fifth day, and died at six P.M.

The causes that disturb the current course of the morbid phenomena have not yet been satisfactorily investigated. They may be due, perhaps, to sudden formation of some poisonous compound in the blood, or the hæmorrhagic yielding of the bloodvessels. Some modes of death may be purely accidental: thus Juan de Susa died from rupture of the spleen, caused by jumping out of the window while delirious. The essential modes of death are modified by those inflammatory complications to which yellow fever is so liable in its course. Thus, M'Kechnie (Seaman's Hospital, 30th of January, 1853) became delirious from evidently neither uræmia nor hyperæmia, but sympathetic suffering arising from pericarditis. Milne's (Seaman's Hospital, 28th of February, 1853) symptoms were modified by an atrophied heart. Devine (Seaman's Hospital, 17th of November, 1852) died with gangrenous lymphatitis; and Savage (17th of November, 1852) died from the shock and pain of acute lymphatitis. The hospital case books are perhaps more deficient in illustration of the mode of death, than of any other of the phenomena of yellow fever. If the death did not occur about the hour at which the reports were written, the final symptoms were seldom described, as, except in extraordinary cases, a single daily report only was made. This arose from the inadequacy of the staff of resident surgeons—the reporters—for the wants of the epidemic period.

There are no sufficient materials to authorize an estimate of the natural

mortality of the present epidemic. Many untreated cases were brought into both hospitals, as the case from the *Rowley* (Seaman's Hospital, 8th of August, 1852), or Francesco Pisthano (Colonial Hospital, 7th of May, 1853), presenting all the well-marked characteristics of the disease; and were, I believe, uniformly fatal. But those untreated cases which recovered, if any, would not of course present themselves at the hospital; hence no comparison can be instituted. It would be a most desirable consummation to ascertain the law of mortality of this disease, as a base line by which results of treatment might be compared; and if some honest homœopath, careful in his diagnosis, would tabulate the results of his practice, he would confer an immense benefit on the science of medicine, by determining the true ratio of mortality of untreated cases. I am in possession of only two well-authenticated narratives which can throw any light on this subject, and they refer to the mortality occurring on board of two vessels after their departure from our harbour. The note which records the case of the brig *Atalanta*, I herewith copy in full, although it contains matters not strictly relevant to this branch of the subject.

"This vessel arrived on the 1st of July. She took up her moorings off 'Holmes Stilling,' nearly in the same place where she lay in January (her last voyage), and in which she was perfectly healthy. Her crew consisted of eight hands and the master; total, nine. On the 19th, two men sickened, and were sent to the Seaman's Hospital. Both cases were very violent, and one died. On the 21st, the vessel sailed up the Demerara river, to take in a cargo of wood at Berlyn, about seventy miles up from town. While proceeding up the river on the evening of the 21st, the second mate sickened. He had refused his supper the evening before. The captain gave him 10 × 12 grains of calomel and quinine, and repeated the dose in three hours, and afterwards gave him castor oil; after which he perfectly recovered, and subsequently was left alone in the vessel to take charge of her. On the 23rd, the first mate and the steward and a boy sickened, and the captain repeated on them his former prescription; but the dose was instantly vomited, and not again repeated. On the 26th, the captain finding that the men were still ill, sent them carefully down to town, in the long boat, to be sent to the hospital or to sick lodgings. They did not reach town till the morning of the 28th, before which all three had died with black vomit. On the 27th, the captain fell sick, and one man, but were quickly pulled down to town in the small boat, and arrived before the long boat, and they went to sick lodgings. After this, the last remaining unaffected man sickened, and was brought to town, and died on the 1st of August. The importance, I conceive, of this instance, is in affording some information relative to the value of treatment, and the probable mortality of untreated cases. They were all gravior cases. The three men who got each the dose which was instantly rejected, may be considered as untreated cases, and all died. Of the two admitted to the hospital, one, or fifty per cent., died. Of the four treated in private lodgings, on the same plan of therapeutics as is pursued in the hospital, one died—the last arrival. The captain states that his former crew had been trading here for three years previously; but that the present crew had not before been in the tropics. This, however, I think will not account for the exemption in the first instance, and the excessive malignity in the present. The lines of infection often shift, and the malaria occasionally operates in vortices; for sometimes one vessel will be suffering from fearful sickness and mortality, while another in the neighbourhood, perhaps not two cables' length off, is exempt. The case of the *Atalanta* contrasts strongly with that of the *Camillus*. In February last, the last-named vessel lost five or six of her crew. The sickness commenced on her tenth lay day. She returned here from London in the latter end of June,

took up her station in the same place (furthest tier out in the river, opposite Johnston and Bros Stelling), and on the *same lay day* the yellow fever again broke out. I advised her being unmoored immediately, and anchored further up the river, which was done, and she has suffered much less this voyage. The *Camillus* seemed to lie in the wind-line of *permanent* infection, blowing from off the Blissingen sluice and the slaughter-house. The unfortunate *Bilair* and the *Honor* lay in the same line.—7th August, 1852."

The brig *Sarah*, of North America, after remaining a few days in harbour, proceeded on to Surinam. She left our port towards the end of July, and after rather a tedious voyage, arrived in Surinam early in August, where she was put in quarantine, and taken in charge of the American consul, till he could send to the United States for a crew—the *captain and all the white crew having died* on the passage from Demerara. This intelligence reached Demerara on the 9th of August, and was published in the local newspapers. The vessel had been piloted from Demerara by a Mr. de Vivre, who on his return gave me information substantially the same as that published. The following is my note of it, made at the time:

"To-day, met Captain de Vivre, who returned from Surinam ten days ago, leaving the *Sarah* still there, under the charge of the American consul. The *Sarah* left this port on Friday evening, with nine of a crew—viz., the master and two mates (*white*), a light-coloured man, three negroes, all natives of North America, and two negroes, natives of Hayti, or St. Domingo. There were also three passengers, two of them females, belonging to Demerara, and a gentleman of Surinam; also Captain de Vivre, who acted as pilot (white creole of St. Eustatia; family several generations in the West Indies, and he many years resident in Demerara), and a negro (?) boy, whom he took with him. One of the crew, a St. Domingian, had been to the Seaman's Hospital for some trifling ailment. *All the crew fell sick the same night* on which they sailed hence. The master, two mates, and light-coloured man died on the following Friday, all within eight hours of each other; all with black vomit, but the master with convulsions. The three North American negroes seemed at one time very ill, but recovered, with the St. Domingians. Nobody else was in the slightest degree affected.—6th October, 1852."

The impression is general throughout the colony that the present epidemic is much more intense than was the preceding; and this opinion is countenanced by the fact that several fatal cases have occurred among the white creole population. As far, however, as documentary evidence goes on the subject, a parallel cannot yet be drawn between the past and the present epidemics; for on the former occasion, eighteen months elapsed before the Seaman's Hospital was established, and trustworthy and extensive records kept; and as the beginning of an epidemic is generally its most virulent period, a comparison with the present is not yet admissible. The following table of thirteen months' admissions and deaths is given, with the explanation, that in the public hospitals of Demerara and Essequibo, in both its main departments, no patient, however ill, is refused admission, *if alive* when the conveyance is sent for him, or when brought to the hospital: that in the colonial department many cases are admitted moribund; that in the selection of cases, when the hospitals are crowded, the preference is always given to the gravior case; and that the hospitals are very frequently made the receptacle of the hopeless cases of private practice.

RETURN

Showing the Number of Admissions, Discharges, and Deaths of Yellow Fever in the Colonial and Seaman's Hospitals, from December 1st, 1851, to January 31st, 1853.

	1851. Dec.				1852. Jan.				1852. Feb.				1852. March.				1852. April.				1852. May.				1852. June.				1852. July.				1852. Aug.				1852. Sept.				1852. Oct.				1852. Nov.				1852. Dec.				1853. Jan.			
	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.	Admitted.	Died.	Cured.	Remaining.
Europeans (residents)	...	...	...	...	...	...	...	...	3	2	1	...	...	...	...	...	1	...	...	1	...	...	...	1	1	...	1	1	1	...	1	1	...	...	...	1	...	...	...	1	1	...	1	1	...	...	...	1	...	...	...	1
Madeirians	3	1	2	...	65	26	18	21	111	36	55	44	134	56	60	62	71	37	40	56	63	26	42	51	51	15	23	64	31	8	32	55	33	9	24	55	16	2	13	56	7	1	7	55	20	...	22	53	18	8	49	14	72	21	40	25
Creoles and Barbadians (coloured)	...	...	...	...	3	...	2	1	1	...	1	1	1	1	1	...	...	...	...	...	2	...	2	...	2	...	1	1	5	1	2	3	2	...	4	1	...	...	...	1	1	...	...	2	...	...	1	1	...	...	...	1	...	...	1	...
Aborigines	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
East Indians (Coolies)	...	...	...	...	3	...	...	3	3	1	3	2	7	1	3	5	4	2	1	6	6	2	7	3	5	...	2	6	5	1	5	5	4	1	6	2	4	1	...	5	1	...	2	4	...	...	1	3	...	...	...	3	...	...	3	...
Seamen of shipping	20	2	18	...	86	13	47	26	93	27	64	28	54	12	40	30	69	17	41	41	76	24	60	33	94	24	49	54	90	26	65	53	88	18	65	58	55	17	57	39	93	19	60	53	110	32	65	66	128	19	99	46	186	48	139	45

TOTAL.

	Admitted.	Died.	Cured.	Remaining.
Europeans (residents)	7	2	5	—
Madeirians	698	246	427	25
Creoles and Barbadians (coloured)	17	2	15	—
Aborigines	—	—	—	—
East Indians (Coolies)	42	11	31	—
Seamen of the Shipping	1242	328	869	45

CHAPTER XI.

The first general anatomical fact in reference to the disease under consideration, is the almost universal *bloodiness* of the tissues of the body in a patient who has died from yellow fever, having been previously healthy, and not dying from actual hæmorrhage. *Hyperæmia* does not express correctly the idea of this condition, for the dissection of the body shows not only too much blood, but also blood in the wrong place. It flows out from the sub-cutaneous areolar tissue; the mesentery is loaded with it; the areolar tissue forming the attachments of the windpipe and gullet is bloody; so is the mediastinum and the fat around the kidneys. The intestines appear, externally slate-coloured, or gangrenous-looking through the peritoneum, from congestions and extravasations in the mucous and sub-mucous coat. The pleura costalis participates in the same sanguineous appearance as the connecting tissue of the throat, from the universal hyperæmia and extravasation beneath that membrane. If we look into the trachea, and bronchi, and œsophagus, stomach and intestines, and coverings of the brain, and lining of the bladder, we find a similar condition in some, and it may be in all these surfaces. If we wash away the mucus or blood which obscures the view, we may find the part highly sanguineo-vascular, the capillaries in a state of distension, without breach of continuity; if in the stomach, leashes of them may be seen torn and disorganized; or the part may show structureless unvascular ecchymosis, and dots or wavy lines, or patches of greater or less extent, or splashes, as if red ink had been projected from a pen. The membrane of the stomach shows the most varied hyperæmia; sometimes it is arborescent, as if the arterial twigs were chiefly affected by engorgement. Sometimes it occupies the rugæ and villi in wavy lines; in other cases it is in rude hexagonals, as if the capillaries surrounding the mucous follicles were alone affected. At other times, the predominating appearance is an universal rosiness, or deep claret or purple, as if the sub-mucous tissue had been infiltrated with pigment; but generally, most of these varieties of hyperæmia are blended together. If we examine the parenchyma of the great viscera, a similar condition of *bloodiness* obtains. The kidneys are sometimes ecchymosed below the capsule, and a section of them is *always bloody*. The liver is very frequently in the same condition, and is sometimes enlarged from general engorgement, and softened and friable in spots, as if from broken-down structure. The lungs are often apoplectic, with the interlobular spaces broken and infiltrated, so as to lose all physical appearance of pulmonary tissue, and resemble huge clots of blood. These conditions are generally found in the most dependent parts, but frequently the upper and front part of the lungs and liver, and pelvis of the kidneys, are so affected. The appearance is therefore clearly not hypostatic, although gravitation must exercise some localizing power. Sometimes one viscus may be engorged, and a neighbouring one anæmiated and dry. This relation sometimes exists between the kidneys and liver, as in the cases of Gibney and Morgan (Seaman's Hospital, 21st of September, 1852); hæmorrhage during the disease, and previous anæmia, have a similar modifying effect.

The next general anatomical characteristic is the altered condition of the mucous membranes. In the mouth, œsophagus, stomach, and intestines, it has suffered some serious alteration. The epithelium is peeled off, generally or partially, or the whole depth of the membrane is softened, as if acted on by an alkali, or is eroded through to the sub-mucous coat.

These are the two general facts common to all normal cases, and obvious to any careful observer. Inflammatory diseases seem congenial to the action of the yellow-fever poison; and during the progress of the disease, we have frequently found them supervening as epiphenomena. We have also seen yellow fever apparently excited into action by their presence, and consequently the traces of these accidental complications will occasionally be found in the post-mortem examinations. But the lesions of yellow fever seem to have little or nothing in common with those of simple inflammation, and the only *quasi*-inflammatory condition which seemed a result of the disease was, in some instances, where the capsule of the liver—such as in the case of Ellwood (Seaman's Hospital, 21st of February, 1853)—was red and vascular, and, as if in incipient inflammation, excited apparently by the mechanical distension of the engorged parenchyma. It is likely that the suppurations of the liver, which have occurred as sequelæ of yellow fever, were occasioned by the disorganization of tissue which follows the congestions and ecchymoses before referred to; and that, had Macey lived, he would have suffered from an abscess in the softened part of the liver, as the only mode of reparation which nature could institute.

In the post-mortem notes which follow in the reports of some of the fatal cases, the terms "blood congested" and "bile congested" have been used, and require some explanation. The first term is obvious enough, and means what it says—engorgement with blood; but the latter does not always mean engorgement with bile: it has reference more to colour than any other quality; and when the liver was yellow, of whatever shade, the term "bile congested" was applied to it, in contradistinction to the dark purple or slate colour which indicated hyperæmia. Now, this yellow condition of the *enlarged* liver is not yet satisfactorily understood. It is no doubt true that the liver is found sometimes dry and anæmic, from having been drained by hæmorrhage, or vital or physical determination to some neighbouring viscus; and then the capsule of Glisson, tinged by the bile, communicates the ochre or straw or cream-colour to the whole mass. But where enlargement also is present, with or without anæmia, the explanation is not sufficient. Of what does this yellow or ochre enlargement consist? This point has not yet received sufficient attention. But I have found that, in these cases, a small portion of the parenchyma scraped off and submitted to the microscope, showed an abundance of oil globules. In cases which have terminated fatally after protracted illness or apparent convalescence, the bloody condition of the kidneys has passed away, and the cortex is hypertrophied, and of a dull ochrey colour. This condition seems clearly due to the impaction of the tortuous tubuli uriniferi with the same epithelial and fibrinous (?) matter which constitutes the sediment of the urine; and the presence of this matter and fragments of tube-casts can be demonstrated by the microscope to constitute a part of this yellow hypertrophy. But I have never been able to detect oil globules in the

kidney; and the instance narrated in the post-mortem notes is undoubtedly a fallacy of observation, the oil most likely having been derived from the blade of the knife. The congestion of the kidneys during life seems to have been attended with no irritation; it is signalized only by albuminosity of the urine. With one exception, nothing like diuresis was observed, nor could have taken place without being noticed, till convalescence became established. Neither was there pain in the congested organ but once or twice (Juan de Nolriga, 24th of December, 1852). The pain so often complained of in the loins is lumbar, and in many of the best-marked cases, careful pressure failed in detecting tenderness of the kidneys. The lesion of the lungs was seldom, if ever, attended by cough or pain or râle, or any sign to attract the attention of the patient or physician until the blood extravasation demanded expectoration. On the liver there seemed to have been induced an irritant effect. The suppression of bile in the last stage had always been preceded by an erethism of that organ, as indicated by the copious secretion of bile, independent (as in the case of the master of the *Undine*), although no doubt increased by the action, of the resolvent dose; and there was frequently tenderness of the epigastrium towards the right side early in the disease, and before it could be occasioned by, as it no doubt frequently is in the last stages, the distension of the capsule: the kidneys and lungs, therefore, seemed to suffer passively, while the liver suffered from active congestion. There was not always a perfect correspondence between the lesion of the kidneys and their functional disturbance during life. Occasionally I have noticed the kidneys in an almost apoplectic state, and yet their functions were scarcely interrupted; and, on the other hand, I have been disappointed in the amount of congestion in instances of entire suppression. In the former case, perhaps, the engorgement occupied less the secreting than the ductal tissue; but this point requires much more investigation. In the post-mortem notes, the weight of the several viscera is given. This precision would have been enhanced had the total weight of the body in each case been also given. Still the weights assist in forming an estimate of the condition; but weight does not in all cases represent the proportional degree of congestion:—thus, in Maxwell's case (Seaman's Hospital, 12th of March, 1853), the kidneys seem to have been naturally small; and although their weight was not extraordinary, yet the engorgement was so intense that their shape became altered to globularity. About one hundred dissections were made since the beginning of the epidemic; but the notes of the first series became confused, and are rejected; and several examinations were made of which no record was kept.

CHAPTER XII.

The common, or *gastro-hepatico-renal, form of yellow fever*, with its stage of febrile reaction and unhealthy subsidence, may be protracted far beyond the average duration of fatal cases; as in the case of Milne (Seaman's Hospital, 24th of February, 1853), or the mate of the *Sobrino*, already referred to. These prolongations of life will generally be found associated with free action of the kidneys, although the urine be albuminous. On the other hand, as in the case of Philips (Seaman's Hos-

pital), who had black vomit within forty-eight hours; and Mr. Dod, who on the second day had, with copious bilious vomitings, *bile in the blood, albuminous urine, and black vomit,* the disease, by the intensity of the epidemic cause, and the high susceptibility of all parts of the body, may be hurried on to early and rapid maturity. In the preceding chapters, *choleroid* and *diarrhœal varieties* have been already mentioned. These refer to the modes of accession, and point to varieties in which the tongue and eye symptoms are generally less early and less distinctly marked, and in which early excoriations around the *anus* occur. In these, the intestinal variety, the *cæcum* is found in a condition similar to that of the stomach; and the lower end of the ileum corresponds with the condition of the upper end of the duodenum in normal cases. In fact, in such cases the cæcum removed from the body might be readily mistaken for a yellow fever stomach. About the beginning of February, 1852, the intestinal variety, or modification of this all-pervading disease, was very common. As has just been stated, in these cases the capillary irritation of the tongue, eye, and face, are generally less observable. In attacks on *old residents*, also, and the black and mixed races—negroes, coolies, and mulattoes—these symptoms are also less characteristic; and the redness of the tongue shows itself at first in the fungiform papillæ only. In the case of the master of the *Hindu*, there were scarcely any *primary gastric symptoms*, and the disease seemed at once concentrated on the kidneys. When the nervous centres seem early affected, and the disease is complicated with *alcoholismus*, the cutaneous system also shows little erethism, and the surface is sometimes even pale. On the 21st of November, 1852, W. Greig died in the Seaman's Hospital, without having manifested the external erethism. On the 27th of July, a case, in which *alcoholismus* was not suspected, occurred, with pale cutaneous surface, in Norman (Seaman's Hospital), and which terminated suddenly and unexpectedly with jet-black black vomit. Williams's (Seaman's Hospital, the 23rd of June, 1853) was a similar case, in which were nervous tremors, but in which intemperance was denied. With *anamics*, as might be expected, and among Portuguese immigrants, the surface symptoms were less striking than among natives of northern climates. In the latter, the disease sometimes assumed a scarlatinoid form; as in the case of Thomas Fry (Seaman's Hospital, 19th of May, 1852), whose fauces were rough, with red vascularity; and Thomas Dawson (Seaman's Hospital, 10th of February, 1852), who, in addition, had ulcerated sore throat. Exceptions, however, appeared, and the most intense action on the capillaries of the skin, as before related, was seen in the Portuguese man, Antonio Fernandez. In the cases of Copeland and James Foster (Seaman's Hospital, 8th of March, 1853), the eyes were as red and injected as in the most violent ophthalmia, but without photophobia or lachrymation. Pericarditis, as a complication, seems to obliterate the surface symptoms, and gives a pale, collapsed, exsanguine expression, as in the cases of Mr. Kichner and Moses Cain (Seaman's Hospital). In the *pulmonary form* of the disease there was no difference in the external symptoms from those commonly observed. But unusual heat of the chest sometimes gave early notice of this obscure variety; and uneasiness, jactitation, and heavy breathing, indicated its approach to pulmonary

apoplexy. Auscultation and percussion have not yet been sufficiently applied in the exploration of this condition, and chiefly from the restlessness and impatience of the subject of it. The *cerebral variety*—that which arises from hyperæmia (and not uræmia)—is characterized sometimes by intense pain in the head and disordered mind in the early stages of the disease; and, as in the case of Laird (Seaman's Hospital, 16th of October, 1852), uncontrollable irritability of stomach. In the late stages, typhomania sometimes occurs from congestion and effusion on the brain, as in the case of John Williams (Seaman's Hospital, 1st of September, 1852), and in whom, as usual, no lesion of the intestinal glands could be discovered. There is some difficulty in discriminating between the effects of uræmia and hyperæmia on the brain, as both conditions are seldom pure. Thus, in Laird's case the one followed the other, and was mixed with it. But the typhomania of Mr. Williams was readily distinguishable, by the assistance of collateral symptoms, from the occasional delirium and insensibility of such a case as that of Charles Maxwell (Seaman's Hospital, 10th of March, 1853), who, suffering from uræmic intoxication, sat up in bed, and amused himself in scolding the nurse and every person around him. Yellow fever, as has been observed already, occasionally came into collision with small-pox; and the latter prevailed, and excluded the former, if in the incommutal stages. It was found *mixed* up, both as a primary or secondary affection, with the following diseases:—Pneumonia (single and double), pleuritis, pleuro-pneumonia, pericarditis, meningitis, lymphatitis, delirium tremens, and intermittent fever. Of all its admixtures, the most numerous, it would appear, were those with pneumonia. But there was likely a fallacy on several occasions in regard to this complication. The expectoration from a softened and broken-down lung was, no doubt, frequently mistaken for the rusty expectoration of inflammatory hepatization. It was in the cases complicated with *delirium tremens* that the peculiarity in the conditions of coagulability of the urine was observed. Mr. C., an Irish youth, about six months in the colony, had been threatened with delirium tremens fourteen days before the attack of yellow fever. On the third day of attack the epidemic disease left him, but the delirium tremens then recurred with its most marked characters, and he died two days afterwards. The Ursuline Convent is situated in perhaps the most miasmatic locality of the town. One of the nuns who died of black vomit had three distinct paroxysms of tertian intermittent, the last ending with the fatal attack of yellow fever. As all our patients in the Seaman's Hospital came from the shipping, the focus of infection, it was to be expected that outbreaks of the epidemic disease would occur among patients who had been admitted thence for other ailments. These did happen frequently, and gave rise to a modification of attack, which we have denominated "threatenings." An extract from my note-book of some notices of this form will show what is meant by the term.

"*Threatenings of Yellow Fever.*—We have frequently seen such in the Seaman's Hospital—men admitted with other ailments, and requiring 20 + 21* as intercurrent treatment. Two such cases occurred to-day and yesterday, in the persons of Griffith Thomas and Charles Burton, well marked, and subdued by the dose. These

* For explanation, see page 81.

cases are prescribed for *pro re natâ*, and seldom entered in the case-books. Sometimes these threatenings finally break out into fatal attacks."—10th August, 1852.

"*Threatenings and Outbreaks in Seaman's Hospital.*—John Hooper, admitted with phthisis, ending in severe attack. Frederick Taylor, gonorrhœa, ending in severe attack; two doses required. Walker, gonorrhœa and stricture—fatal. Duncan Cameron, fractured clavicle—one dose. William Cransick, second threatening. William Roberts, threatening."—15th, 19th, 22nd, 24th August, 1852.

"These threatenings are generally marked by flushed face, frontal headache, with considerable warmth of forehead, suffused eyes, and quickened pulse. If not stopped (but they generally are) by one resolvent dose, they are highly dangerous. They frequently occur and are prescribed for *en passant*, but when aborted are not noticed in any of our records. The incubation of some of these attacks seemed retarded in the presence in the system of such evacuant diseases as phthisis and gonorrhœa."—25th August, 1852.

"*Threatenings of Relapse.*—Three cases occurred yesterday in Seaman's Hospital in convalescents from yellow fever, all extinguished by one dose each."—13th September, 1852.

"*Attack while in the Hospital with crushed fingers, repulsed*, twice within a few days, by four powders, in case of Mate Anderson."—Seaman's Hospital, 27th December, 1852.

"*Two Threatenings of Yellow Fever*, one coming with chills, among those some time in (Clinch and Freeman), aborted by one dose each. This happened yesterday; close, warm, humid weather, followed by torrents of rain to-day."—18th May, 1853.

There was a form of the epidemic disease which was known in our case books by the name of "smouldering." This type is distinguished by the absence of any rampant symptom. It seems to be a *non-localized* variety, or so equally but mildly spread over the system that no organ is intolerably implicated. This variety *begins* in much the same manner as the common form; but if the attack be not aborted, it proceeds differently. Its peculiarities begin from about the second day. In its mildest degree, the disease then seems to have nearly departed, but the patient remains languid. The skin, which is nearly cool, becomes purplish over the face, arms, and chest chiefly (languid capillary circulation, as it is called in the case books). The eye gets tinged, the urine is free but bilious, and, for a day or two, albuminous. But gradual restoration to health follows. In severer cases these symptoms are all intensified, but still the kidneys are not much involved; the stomach remains quiet; there is little capillary irritation of the mucous membranes visible, and little or no peeling of the epithelial covering of the tongue. But the skin has a macerated, greasy appearance, and beads of acid perspiration stand on the forehead, on the alæ of the nose, and around the mouth, and sudamina appear over the body. Around the anus and over the lower surface of the scrotum, the cuticle peels off; and if any blister has been applied, the surface there is of a claret colour. The blood is alkaline, and the matter of the excoriation also. A small bloody anthrax appears (in the case I have now in view—the mate of the *Alexander Johnston*, private lodgings) on the left elbow. The patient throughout has been and remains taciturn and apathetic, and speaks in a subdued, low tone of voice. This brings his case to the end of the third day. Incipient black vomit may appear, as it did in the mate's case on the fifth day. Convalescence then begins, but is slow. The junction of the gums and teeth oozes blood, which stains the fur of the tongue, as if with tobacco-juice; but the appetite is good; the skin clears between

the interstiees of the sudamina; these little vesicles dry up and desquamate; the powerful action of the kidneys soon relieves the circulation of bile and decolours the eye, and health is perfectly restored. Few good descriptive eases of this form of the disease are to be found in the case-books, owing to the circumstance that there are so few salient points to be described, and little indication for any interference with nature on the part of the physician. Such cases, in the hospital reports, break off, generally at the second or third entry, with such a prescription as the following:—℞. Aq. acet. ammon., aq. camphorat., āā. ℥ss. ter in die; and no report is again made until eight or ten days after, when he is marked discharged; the medicine in the meantime having been omitted, probably after two or three days' use. Of such cases were England (Seaman's Hospital, 19th of July, 1852), Lamont, Dixon, Pullin, Jenkins, Russell, Gorman, Forsyth, and W. Davies (Seaman's Hospital, December, 1852). The pathological rationale of this variety of yellow fever is yet to be cleared up; but it would seem as if the less vital structures were the throne of the disease in such cases; that it occupied chiefly the periphery of the body—the cutaneous capillaries instead of those of the central viscera. I am also inclined to believe that, though diarrhœa has not been found to usher in the attack, it is sometimes the manifestation of the *intestinal* variety, or in alliance with it.

Akin to this form of the disease is another. It was noticed in the last epidemic. It appears most frequently among the dark races, and some of the Madeirians. It is, perhaps, a mere extension of the *smouldering* form, but the symptoms are more typhoid. It has strong resemblances to sea scurvy, if we could conceive that to be an acute disease, and hence may be named the *scorbutic variety*. It is generally unattended with gastric symptoms, nausea, or vomiting; and instead of the raw-beefy, clean, epithelium-denuded tongue of normal cases, the mouth seems as if smeared with tar. The following note, which I made in my memorandum-book for another object—viz., to illustrate the *formative* stage of yellow fever—will describe the scorbutic variety in an European—not pure, certainly, but sufficiently characteristic. Although this case was eminently suggestive of other important matters, the phenomena at the time were not duly appreciated nor correctly interpreted, and it was regarded only as illustrative of the mode of invasion of yellow fever.

"*Formative Stage of Yellow Fever, showing itself by Boils on Forehead.*—Captain Sutherland, of the brig *Hero*, Shetlander, a very tall, narrow-chested, and very old-looking man (although it is stated that he is only forty-five years of age), got yellow fever, in the invasion stage, on the night of the 18th instant. I had seen him every day for a week previously, while I was attending his mate for an attack of the gravior form of the epidemic. For five or six days before the invasion his forehead was covered with little numerous unhealthy boils, which prevented him from being able to wear his hat. As will be seen by the sequel, this was evidently the first manifestation of a peculiar case of yellow fever, closely allied to scurvy. The febrile symptoms were low all along while they lasted (two days). While under them, he walked along a plank ashore to meet me (the brig lay alongside the wharf, and the mate, who recovered afterwards, lay sick in an adjoining house), to save me the trouble of going on board. He was with difficulty persuaded to lie up. When the short, trifling fever left him, his pulse fell much in power (particularly observed in right radial artery, probably from a natural difference of size in the artery), and became very slow—forty-eight. The chief symptom, and which persisted, was a

crimson-tipped tongue, wedge-shaped, and the surface behind tip foul and brown, and inclined to dryness. His gums began to bleed and recede from the teeth. He hawked up rusty sputa from mouth and fauces. His restlessness was easily, and *much too easily*, quieted by a few drops of morphine solution. In fact, this is the first case of the present epidemic in which morphine was detrimental. The boils on the forehead began to fade and become purplish. Other and larger boils, with an unhealthy dark-purple centre and apex, and coppery inflamed areola, came out over arm, body, and legs; and some bullæ, which burst and bled dark grumous blood. Anorexia; extreme faintness and nausea on assuming the erect position ensued. Stomach easily irritated by the swallowing of food and drink, but never decided vomiting. Feebleness of voice; urine copious and dark-coloured. Bowels inclined to torpidity, but easily acted on. To-day, two of the boils on right leg bled much; so much, that I was called to arrest the hæmorrhage. They looked livid and much swollen from a large black clot, which I tried to remove without using much force, but could not. I think it had the appearance which is described as 'bullock's liver' in sea scurvy. A little stream of red blood trickled out from the clots, which lead me to suspect that a small artery had given way. Hanging down the leg or attempting to stand made the hæmorrhage, which otherwise oozed, alarming. He to-day also complained of much *pain in the side* (the dangerous sign in sea scurvy, described by Lind) below the right axilla, midway in side; and from an imperfectly-made auscultation with the ear, dulness of respiration was found over seat of pain, and an occasional coarse crepitus. He now also *expectorated* rusty tenacious mucus. A dry cup was applied over the seat of pain, with some relief. Notwithstanding the most liberal and varied use of cordials, and soups, and anti-scorbutics, he sunk during the day, rapidly, and when I last saw him—about half-past 7 P.M. to-day—he was restless and moaning, and almost pulseless, and unable to ascribe his uneasiness to any local pain. No vomiting or nausea. His breath and body have been exhaling a disagreeable, sour (?), fetid smell all day. I do not expect to find him alive to-morrow, 25th of February, 1852.

"I may mention that, in the foregoing case, I examined his legs to-day, to detect that hardness of the muscles which authors say is found in the legs of those who suffer from sea scurvy. But the muscles and integuments were quite flaccid. He never had been on a very long sea voyage, nor suffered at any time from sea scurvy, and has been in this harbour nearly three months. Does the foregoing case not seem to show that, in yellow fever, the blood is primarily affected? 25th of February, 1852.

"Captain Sutherland died at midnight; his urine was free; he continued moaning, but his intelligence was clear." 26th of February, 1852.

By Peter Daley's case, before referred to, the notion that hæmorrhages of yellow fever have their origin in a dissolved state of the blood, was staggered, and a lesion of the solids looked to, with probability, as at least a joint cause. This modification of opinion was for some time unsettled, and the dubious and unsatisfactory phrase of "loss of vital cohesion," all the explanation applicable to the condition of the arterial tubes which the mind had to lean on. The case of Captain Sutherland is now much more intelligible than at the time when the foregoing notes were written. It was clearly a combination of the scorbutic and pulmonary forms of yellow fever. But so far do I now consider it from proving that the disease is primarily a blood affection, that I feel convinced that the scorbutic variety arises from the yellow fever poison acting chiefly on the *arterial twigs* and capillaries. If this view be correct, it might be well for those who have the opportunity, to re-examine the current pathological opinions on sea scurvy.

It will have been perceived, that the distinctions of variety and form in yellow fever are to a considerable extent artificial; that one variety is seldom seen pure; that they are generally mixed and blended. Still, the distinctions are not without differences, and they assist in understanding the malady in its Protean shapes. The *simplex form* of the last epidemic was not observed in the present, in the coasts and low lands. Neither was it observed in the last till the epidemic had continued upwards of two years.

CHAPTER XIII.

On the subject of diagnosis and prognosis much has been necessarily anticipated in the foregoing observations. I know of no disease with which a well-developed case of yellow fever could be confounded; and to those who understand its habits it can rarely present any serious difficulty of diagnosis in any of its stages and complications. In the early stage alone can a mistake be made. The absence, then, of some of the external symptoms should induce the practitioner to extend his inquiries, and he will rarely find a case in which the capillary irritation is not observable on some part of the exposed mucous surfaces. Should such an instance occur, he will find the fever accompanied by that peculiar form of headache which is decisively diagnostic. This headache gave the earliest warning, and in Surinam and Cayenne was relied on as the most characteristic symptom. Should the capillary irritation not appear after the lapse of forty-eight hours, then he must look out for those complications which have been already indicated—the chief of which is pericarditis. I could conceive much embarrassment on the part of the practitioner in distinguishing yellow fever from benign scarlatina, if the two should happen to co-exist as epidemics; and I am quite unprepared, at present, to assign any satisfactory differential distinction. But when the stage of acid elimination has been marked, all doubt must then be at an end, for this phenomenon is unknown in any other fever. I have seen instances in which yellow fever was stupidly mistaken for ophthalmia, and the disease treated accordingly; but not in the present epidemic has such a blunder come to my knowledge. With the mind of the practitioner alert to the existence of the epidemic constitution, he can, by intelligible signs, recognise even the taint which is communicated to other maladies, and identify the disease, with moral certainty, amidst any of its complications.

On the 3rd of January, 1853, Manuel d'Alrea presented himself for admission to the Colonial Hospital among the crowd of other applicants, and complained only of pain in his side, cough, and fever. On applying the ear to the affected part, pleuro-pneumonia was readily discovered. But on observing afterwards the state of his tongue, the presence of yellow fever was at once recognised. On further examination of the urine, the same day, it was found highly albuminous, and the existence of the double disease was confirmed by the sequel.

On the 29th of June, 1852, an exactly similar case had occurred in Vincent Gomes, in which pleuro-pneumonia was the primary disease, and fatal yellow fever consecutive, but detected in its onset, and through the disguises of the leading affection, by the tongue symptom.

I have had no opportunity since it became important to ascertain the

fact, of testing the urine in that intensified form of marsh or malarial fever commonly known as *bilious remittent*, and locally with us, *colony fever*, and cannot say if albumen be present or not in any stage of it. But numerous observations have failed in detecting it in the base or radical of that disease, intermittent fever: and the capillary irritation and acid elimination during life, and the altered condition of the mucous membrane of the alimentary canal after death, are, I believe, never found in bilious remittent. In this latter affection the mode of death, with us, is generally by coma, and the sub-arachnoid effusion and opaque arachnoid membrane explain the *rationale* of it. In it the spleen and liver may be enlarged, but the kidneys are unaffected; and, save the jaundiced tinge, the traces of diseased action, such as in yellow fever, are not seen. When vomiting occurs, it is when the congestive stage has reached its acme at the termination of the chill, and, except in drunkards, rarely extends into the hot stage. Engorgement of the liver and spleen and portal circulation, and not irritation of the mucous membrane of the stomach, seem to originate the vomiting, and be relieved by the copious discharges of the bile that follow. The headache is in the temples or top of the head, or all over it. The stomach, instead of becoming more irritable, is settled as the disease advances, and large draughts of fluid are easily retained. Calomel and antimonial powders, in small and frequently repeated doses, act soothingly. The teeth and tongue are dry, but not red and vascular, and the epithelium apparently worn off at the tip, as if by attrition with the teeth. But the epithelium in reality is only *shrivelled*, and when the remission occurs, the tongue is flat, moist, and pale, and its epithelium is found entire.

Yellow fever, although it may be engrafted on an intermittent, when once formed has no intermissions. It is a fever of one paroxysm, without the crisis of perspiration; and when it is over, health is restored, or the disease goes on inducing its ultimate changes without febrile action. The time of seizure is different with yellow fever from that of our permanently endemic fevers. It generally comes on in the night half of the twenty-four hours; while with us, all our miasmatic fevers, whether quotidian, double quotidian, or tertian, in the immense majority of instances, occur at mid-day. And, if we follow intermittent into its sequelæ, we find no resemblance between the two diseases. There is not the quick restoration of health usual in yellow fever, nor the bloody furuncles of unhealthy convalescence; but instead, enlarged spleen, anæmia, dropsy, and colliquative dysentery.

Although frontal as well as general headache, with lumbar and other muscular pains, usher in the fever of small-pox, the absence of gastric irritation and capillary injection of tongue, lips, and eye, is sufficiently distinctive.

The number of the characteristic symptoms present, and the degree in which they are manifested, furnish criteria of the severity of the case, and the ratio of danger. A slow pulse and moderate temperature of the body and quiet stomach are always favourable indications. But the more fiery crimson the tip and edge of the tongue, the more irritable the stomach, the severer the headache, the worse the prognosis of the first stage, and *vice versâ*. Slight or moderate epistaxis is a sign of little

prognostic value in any stage; but a streak of blood in the early vomit indicates much danger from the attack; while the same during the stage of black vomit, or after acid elimination has set in, is favourable, if the corpuscles are found entire. In the second stage, the earlier or more complete the suppression of urine and the more copious the ejections of black vomit, the more imminent the danger. But if the urinary secretion continue, and the black vomit be scanty from the first, or is afterwards suppressed, the patient may yet survive. Urine simply albuminous is a less serious sign than when it also contains tube casts; but if these are thin and few in number, they do not add much to the gravity of the indication. Free, copious urine, no matter how dark or bilious, is the most favourable of any single sign. If the urine be scanty, and it be loaded with tube casts, entangled in epithelial and fibrinous (?) matter, the light buff-coloured curdy sediment before mentioned, it indicates a complex lesion of the secreting structure of the kidney. It is the urine symptom in its maximum of severity, and is as fatal as if the suppression had already occurred. Blood corpuscles in the urine were not looked on with apprehension. A faltering of the articulation is a bad prognostic, and a difficulty of protruding the tongue enhances it. Prognostics are derived from the effects of treatment. If the resolvent dose do not bring away "stools characteristic of the powder," but, instead, thin grey abilious matter; or if early hypercinchonism be induced, it is an unfavourable indication. The danger of the case is enhanced by inflammatory complications, and by hypertrophy of the heart. The recency of residence in a temperate climate; the *race* or complexion of the individual; the fact of his previously having suffered from a gravior attack, or an aborted one, will enter into an estimate of his chances of recovery. It is unnecessary to recapitulate the modes of death. These are signs too late to be of any practical importance.

CHAPTER XIV.

Till the 6th of January, 1852, the profession and the public were unaware of the presence of yellow fever in the community. The fatal cases in the family of Mr. Vervestein had not been recognised by the practitioner in attendance (who had never before seen the disease), and nothing was said of them till after the diselosures from the hospitals had been made to the local government. Dr. Gavin, the Medical Inspector for the West Indies, then in the colony, learned the fact from the government secretary's office. This circumstance is mentioned to demonstrate the difficulty of discovering *first cases* in any epidemic, unless where the opportunities of observation are as ample as the vigilance is unceasing. The manner in which the epidemic invasion commenced in the shipping, is described in the official communication to Governor Barkly, of the foregoing date, how it began by tainting the ordinary endemic fevers, and gradually acquiring intensity, till the disease became a well-developed primary affection. This communication and the appended documents, as well as a statement of the health and meteorology of the non-epidemic period, have already appeared in the third edition of the "Account of the Last Yellow Fever Epidemic of British Guiana." The particulars in regard to the two fatal cases therein referred to, as the earliest in private

practice, and occurring about the 22nd of December previously, are as follows:—Mr. Vervestein, an Englishman by birth, thirty-two years old, twenty-eight of which he resided in Barbadoes, took up his residence in Georgetown, Demerara, about two years prior to the outbreak of the epidemic. His domicile was in Carmichael-street, opposite the south-west end of the parade ground, and close to, and directly to leeward of, what was then a very wide, putrid, offensive trench. Mrs. Vervestein and three children rejoined Mr. Vervestein from Barbadoes, exactly three months before the first case of sickness appeared in the family. The children, on arrival in the colony, were remarkably plump, ruddy, and clear in complexion, as if they had arrived from England, instead of the West India Islands. The first case was in the person of his son, five years old, who recovered. Then, and while his brother was sick, a younger son, three years old, sickened and died. Then the father sickened, but recovered. And lastly, the infant, eighteen months old, sickened and died. The two deaths occurred within a week. The mother remained quite healthy. Mr. Vervestein informed me that his father died in Barbadoes, after residing there several years, of yellow fever, at the age of sixty-three. This family had no connexion with any source of human contagion; and the cases which immediately followed them, and the cases which occurred at several intervals of time in the same street—viz., the governor's white maid-servant, Mrs. S., Mrs. B. H., and Mrs. H.'s white maid-servant, had no communication with each other; and except in the Vervestein family, each case occurred in a single form, and without any lateral offshoots.

From the statements already given of the progress of the epidemic from east to west along the coast of South America, it has in this instance every appearance of having been an *imported disease*, though not in the sense usual to that term. The condition of the coast line, which had been observed as coincident with former epidemics, was in this instance absent. The epidemic outbreak also recurred in a much shorter cycle than we had reason to expect from past experience; and however much it might be domiciled, and sustained, and reproduced (for though the pestilence extended on to leeward, it at the same time remained with us), a comprehensive view of its whole march enforces the conviction, that in this instance the prime exciting cause had its origin beyond the bounds of the colony.

It would appear from the observation of the present epidemic that though, as is well established, a certain high average temperature is required for the generation and continued existence of the efficient cause of yellow fever, it has not its genesis from any known combination of meteorological elements, and may appear at a time when they are highly favourable to general health and comfort: that the laws of its diffusion differ from those of gases: that it is impelled by atmospheric currents, but seems to possess some power of spontaneous motion: that though intense energy of vegetative power characterized the seasons antecedent to and during the epidemic invasion, its shifting lines of infection and gyratory movements suggest to the imagination the attributes of insect life: that the development of its power was gradual, from its feeble and diluted manifestation at the end of October till its perfectedness at the

end of December, and its maximum of intensity a month afterwards: that during the course of its progress it showed marked variations of epidemic power: that in constitutions apparently the same, the system was affected in various degrees, as if the poison acted in proportion to its quantity, and as a poison and not a ferment: that its first impression on the system seemed in many cases local and circumscribed, although attended with the usual constitutional disturbance: that it can actively occupy the body simultaneously with other affections, and may be either subordinate or paramount in the issue: that though its extensive application or saturation of the system by the efficient cause eventuates in a spontaneous outbreak of the disease in the individual, there are circumstances which accelerate its action and augment its intensity, and others which retard or entirely obviate and render it inert. These circumstances will now be considered, and though little that is new can be added to the subject of aggravating, exciting, determining, and predisposing causes, and the conditions of comparative immunity, the fresh illustrations may be useful in corroborating former experience.

As to the effects of personal *contact*, mediate or immediate, with those sick of the disease, the case of the brig *Sarah* has been already noticed, in which all the crew sickened simultaneously, and all those of the white or mixed race died within a week. The following is the newspaper article which then appeared, and the statements of which were subsequently confirmed. The italicising of some of the words is mine:—

"*Paramaraibo*, 3rd Aug., 1852.—Last week arrived at Braams Point the North American brig *Sarah*, Captain L. S. Griffin, from Demerara, which, in consequence of the sickness on board, was put into quarantine. His Excellency the Governor immediately ordered the health-officer on board, for the purpose of treating the sick. According to the reports sent in by the doctor to yesterday's date, we learn the death of the captain, first mate, second mate, and one sailor; but fortunately the others had all recovered. We understand that the passengers, *who remained throughout healthy*, will in a few days be allowed to come to town."

Mrs. Vervestein, who must have been in the most intimate contact with the infant who died in her family, alone escaped an attack. In the book of the Colonial Hospital, it is mentioned in the last epidemic that a Maltese woman, named Fannia, died of yellow fever on the 16th of January, 1840, and while she had black vomit, nursed her infant at the breast, without communicating disease to it. The brig *St. Fillan* lost three men in Demerara with yellow fever in the present epidemic, after which she proceeded to Berbice to take in cargo. Immediately after her arrival there, two of her men and the mate sickened and died. They had obviously contracted the disease in our port, and transplanted it to the neighbouring country. But it did not grow. No steps were taken to prevent the spread of contagion, if any existed, but the importation was without issue.

We had two specimens of the West India Island type of the disease imported into Demerara while our epidemic was in progress. Mr. C. T. Chandler and his brother arrived on the evening of Tuesday, the 28th of October, to take shipping for England on their way to Australia; the age of the former was about twenty-two; he was a white, a native of Barbadoes, and had never before been out of that island. He had sickened with yellow

fever that there then prevailed, on his passage over. I saw him on Thursday evening for the first time; the brother not alarmed, supposing his illness to have been only sea sickness and its effects. He died with black vomit, on Saturday morning. I watched the influence which this case might have on the health of the inmates of the house in which he died till the 11th of December following, but no effect could be observed.

Mr. B., a passenger from England by the mail steamer, at the end of December, 1852, contracted yellow fever while the steamer lay at some of the infected islands—probably Barbadoes. It was four days old when he arrived in Demerara. A bloody furuncle formed on his cheek during convalescence. No injurious effect followed either to the inmates of the house or his friends, who had the most unrestricted communication with him.

The *Lancaster*, *Atalanta*, and *Flirt*, proceeded up the Demerara river to load with timber, at different times, after having been some time in the port. These vessels lost while there more than half their crews. There is a population of about 500 where they loaded, about fifty of them whites. These people bring the timber, and assist on board the vessels, and the most unreserved communication is kept up between them and the sailors. They were constantly going and coming, and the sick and dead were landed among them. But not a single case of yellow fever occurred among that population.

No case occurred spontaneously in the district of Mahaiconey; but Mrs. M., who resided there, contracted the disease in town, and died after her return home with black vomit. The disease did not spread. Private lodgings, during the epidemic, were in great demand for the ship officers and many of the seamen. In a memorandum of the 17th of January, 1853, I read as follows:

"*Number of Yellow Fever Patients in Private Lodgings.*—Yesterday, when visiting Mrs. Morison, wife of the master of the brig *Hope*, of Carrickfergus, Mrs. Fraser (white), who keeps the lodging-house, brought to my recollection that Mrs. Morison was the forty-second case of yellow fever which she had had in her house, of whom I had attended all but four. I have no doubt but that Mrs. Thompson (mulatto), at the corner of Main-street and Regent-street, had twice as many in her lodging-house; that Mrs. Wood (mestizo), had as many as Mrs. Fraser; that Mrs. Hobbs (mulatto), in Robb-street, had also as many; Mrs. Frances Porter (mestizo), in Water-street, had as many. Besides these, other houses took in sick seamen—such as Miss Catherine Mortimer (negro), and Mrs. Milleman (mestizo). Among them all, however, not a single instance of contagion, or any suspicion of it, has ever arisen. Nor have they, nor their servants, nor visitors, nor washers, furnished a single case of the epidemic disease."

On the 1st of January I find the following entry among my memoranda:

"*Number of Servants in the Hospitals.*—In the colonial branch there are forty-three constantly employed, and frequently changed. There are nine in the seaman's department, constantly employed and occasionally changed. Total, fifty-two constantly employed. Of these, since the commencement of the epidemic, two have suffered from yellow fever—viz., Maria de Monte, who is in charge of the Lazaretto, and is never near the fever patients, and whose case was complicated with pneumonia; and Manuel, the Portuguese interpreter, who never does duty as nurse. A nurse named Caruthers was laid up two days with a 'threaten-

ing,' and the head cook (mulatto), while suffering from orchitis, showed some taint of the epidemic. The number of servants stated is exclusive of those employed in clothes washing. The washing is done by contract, and none of those engaged have suffered. The servants employed about the hospital are Madeirians, negroes, mulattoes, Europeans, and coolies, in about equal numbers."

In the fatal cases which have occurred among the white natives, and excited the notice and consternation of the community, and who during their illness had "troops of friends" around them, not a single instance of contagion was suspected to have happened. Having watched, however, for instances (it being easiest to look after and record exceptional cases), I discovered among them one, which may be open to suspicion till the circumstances are explained. Mrs. W., white, native, aged about twenty-five; four or five years without having left the colony, sickened and died on the 17th of January, 1853. Her residence was a considerable distance from Water-street, in a hitherto healthy locality, on the Brickdam-street. Exciting cause of attack unexplained. Her maid-servant, English, elderly, four or five years in colony, had been very assiduous in her attentions to her mistress, but not more so than some of the relatives of the deceased. She, however, sickened, and had black vomit *incipiens* on the 28th of February following, but recovered. At the same time, the son of the deceased, two years and half old, sickened and died. Though the symptoms were somewhat obscure, and the diagnosis unsatisfactory, and the disease ended in convulsions (an infantile affection then prevalent), yet there can be little doubt that the cause of death was yellow fever. Neither the father, nor two elder children, nor son of the nurse (English and about ten years old), nor any other inmate of the house, suffered. The grouping of disease in this family was entirely exceptional among cases affecting *white natives*, for in all the other instances the attacks were isolated and singular. Now, although the distance of time between the first and second cases in this family is considerable, still there is an appearance of probability in the idea that the disease had been communicated from the mother to the nurse and child, until we know the history of the other cases in the neighbourhood. At this time the poisonous atmosphere had evidently extended to the Brickdam, for a fatal case in the person of a Scotch lad, a few months in the colony, followed that of Mrs. W. exactly a month afterwards, and in the next, but *separate* and *windward* dwelling, though between the houses and inmates no intercourse whatever had taken place. On the 24th of February also, about two hundred yards further up in the same street to windward, a second attack of the epidemic, in the person of Master B., occurred; and on the 25th there was another seizure, in the person of Miss S. (mestizo), about one hundred and fifty yards to the south. Both the latter were aborted attacks. Now, it is certain that none of these last-named cases had any personal communication with each other previous to the advent of their respective attacks, and they appeared *successively to windward*, until the nurse's seizure again manifested the infection in the old locality. Although the nurse and child, who remained in the house with the mother, contracted the disease, none of the friends from a distance who visited and closely attended the sick, suffered on their return home, but from fatigue.

There is the most unreserved intercourse between the ship masters and those connected with the public buildings, particularly of the custom-house chambers. All the public offices are in the one extensive building. Of officers permanently employed in this building there are sixty-three, and only two or three of them "coloured." Nine officers are in the custom-house. Two only of the whole number were affected, and these two were lads lately from Europe, and not in the custom-house, but the opposite end of the building, the registrar's office. Between these two cases there was an interval of several months; one recovered and one died. They were treated in the midst of their respective families (whites), without restraint or hindrance, and without any sign of contagion ensuing. On the other hand, Dr. Levin, resident surgeon of the hospitals, died on the 1st of May, after three days' illness, of the prevailing disease. He was a native of Russian Poland. He had been five years in the colony. The exciting cause of his attack seemed to have been fatigue and exposure to a thorough draught when violently heated by the pursuit of a thief whom he detected stealing his property. He also had been previously in the habit of passing the evenings in Robb and Water Streets, in the main site of infection. Drs. Butts and Goring, resident surgeons, each had an aborted attack; but both had lately arrived from cold climates, England and Canada. As already mentioned, many cases of yellow fever appeared within the Seaman's Hospital, in the persons of patients who had been admitted for other ailments; but they had all been exposed to the river influence previously. As the object of our observations was to ascertain the truth, and hold by it wherever it might lead, these cases were watched as sedulously as any partisan of contagion in yellow fever could desire. Among them, only two cases of an equivocal character appeared, and which bore favourably on the doctrine of contagion. They are as follows: George Philips, of the *Harkaway*, was admitted three days after his arrival in port to the Seaman's Hospital for rheumatic ophthalmia, on the 26th of February, 1853. On the 5th of March following, he was suddenly seized with a violent attack of yellow fever. As usual, there had been no separation between his and the yellow fever cases. Now the only suspicious point in this case, as regards contagion, is the fact that up to that period the *Harkaway* had as yet furnished no other case of yellow fever that I could ascertain. The other case was that of William Smith, of the *Montezuma*. These cases occurred very close on each other. After having been one day in harbour, he was admitted to the Seaman's Hospital for bubo, and was literally covered with *acne punctata*. On the 7th of March, after mixing as usual with the yellow fever patients, he was seized with what proved a fatal attack. In this case also none of his messmates had as yet suffered from the epidemic.

I have now stated all the facts that have come to my knowledge during the course of the epidemic, which favour the doctrine of the personal transmissibility of yellow fever. They were earnestly looked for among the countless opportunities for observation, and no others could be discovered. Those which were found have been honestly declared. In such a poverty of positive proof in the affirmative of the doctrine, it is no argument against those who disbelieve in the doctrine of contagion, to

assert that their proofs amount to negative evidence only. The experience of the present epidemic has confirmed that of the past, and the idea of contagion, which was then unanimously relinquished, has not been revived. Neither do facts countenance the fanciful compromise which some have offered as a settlement of what is scarcely a question among those who in modern times have seen the disease with their own eyes—viz., that it is *the type of disease in which black vomit appears only which is contagious.* In Demerara we would as soon think of asserting that intermittent fever in some of its forms and types is contagious, as to predicate it of any of the manifestations of yellow fever. It has already been observed that the state of the weather exercised a modifying influence on the manifestations of the epidemic. Heavy rains, with calms, creating a damp, hot, steamy atmosphere, or the prevalence of land winds, which are cold (comparatively), damp, and of low dynamic power, intensified the action of the poison, augmented the number of admissions, and increased the severity of the symptoms. The return of a dry, cool, clear, elastic atmosphere, with sweeping trade winds from the ocean, was always followed by mitigating effects. The *rationale* is easy. The condition of the weather first referred to oppresses the cutaneous and pulmonary functions, and thereby lowers the tone of health, and its power of resistance to the action of noxious agents, at the same time that the stagnation of the air is favourable for the accumulation of the atmospheric poison, whatever it may be. Moreover, it is likely that, at such times when the wind is from the land, and the sky is darkened (as it always is when land winds prevail) by dense black clouds, which overspread it from zenith to horizon, the air is positively vitiated by an excess of carbonic acid gas. The whole country is thickly covered by the most luxuriant foliage of grass, shrub, and tree (rarely fleshy leaved), the purifying influence of which on the atmosphere must be impaired during the temporary diminution of light. Whether during the darkened stagnant state of the air which then exists, the vivifying supply of oxygen be lessened, or the amount of carbonic acid be increased, effects detrimental to the vital force must ensue, which would be favourable to the ravages of the epidemic. It arrived on our shores at the latter end of the year, between the autumnal and vernal equinoxes, when the trade wind blows day and night over the face of the country, and it may have been owing to this accident—anachronism—that the invasion was so feebly commenced, and required so long a time to muster its forces.

The most important influence that could be brought to bear against the susceptible, was that of locality. As in the former epidemic, and as already noticed, the focus of infection was the shipping and Water-street. The very same houses that before signally suffered, were again visited with a like severity. The poisonous agent persists in its predilection for low, damp, crowded places, and putrid exhalations, and woe to the unwary or reckless who lived or lingered there. In the house next to that which Mr. Vervestein occupied, Mrs. B. died about two months after the first cases. Two tenants who successively occupied that house also suffered, but recovered. Persons who on business or for change of air came to town from the uplands of the interior, suffered. The Rev. Mr. L., safe at the missionary station near the penal settlement, came to town

on a visit, and died. Mr. Charpentier, from a more remote position in the Upper Essequibo, brought his family to town for the sake of coast air, they having suffered from intermittent fever, anæmia, and enlarged spleen, in the interior; two died with black vomit. Vessels that carried coal in bulk or patent fuel, were severely visited. The history of the *Syrophœnician* and *City of Peterborough*, and several other instances, illustrate this point. I have been informed of the case of a Portuguese boy, who appears to have contracted yellow fever by being much on board the *Grafton*, while discharging coals at Plantation Houston, at a time when the crew also suffered. The plantation was at the time healthy, and remained so till long after any influence could have been exercised by the presence of the sick boy, and between whose case and those that followed, no connexion could be traced.

Fatigue and checked perspiration and long-continued solar exposure precipitated the attack. Sometimes the heat of the berths below forced the officers to get up on deck at night, where they sat or lay with little covering to either feet or body; and a chill following was the signal of seizure. The *tolerance* of the poison which those residents who passed through the epidemic from its first feeble manifestations, had acquired, was seriously impaired by even a temporary removal from the colony, and a return to it within a few weeks. The crews of East India ships were severely visited. It might have been from the nature of the previous cargo, rice and immigrants, or from the long voyage inducing in the crew a scorbutic diathesis favourable to the reception of the epidemic poison. The depressing emotions of the mind were highly favourable to the action of the poison. Worry and vexation, crushing sorrow, panic, and even overwhelming joy, have each had its victim. Among the shipping, when the disease began, panic multiplied it; and the same emotion, in the open wards of the hospital, no doubt swelled the mortality. Constant and brisk employment, under awning, was the best prophylactic for the seamen. A week's idleness, which enabled them to gossip over the exaggerated tales of sickness and death, was enough to start the infection. The boy Lawrence (Seaman's Hospital, 11th of June, 1852) was convalescent, and in the morning of the day of his fatal relapse, was laughing in the convalescent ward. He was moved from his bed, which was sheltered from the wind, to make place for a bronchitic patient. He mistook the motive, became alarmed, wept and sobbed, and was not to be pacified; was seized with fever immediately; demanded to be sent back to us (the acute case ward), where he died. One of the most singular instances of what could only be accounted for by the effects of moral emotion in the genesis of yellow fever, occurred at her Majesty's penal settlement, in the high lands of Essequibo. A convict, native of Madeira, who had been imprisoned there for eighteen months, died on the 29th of July, 1852, of a well-developed attack; a minute and very interesting account of which was furnished me by Dr. Ringer, the resident surgeon of the settlement. Till then, no case of the epidemic had been seen or heard of in the uplands of the interior, and none followed till November, when the *simplex form* ran through the whole settlement, but without a single death. On the 17th of August, 1852, Dr. Ringer writes—"I am happy to say that this is the

only case that has occurred here; and lately we have had below the average amount of intermittents, and, with the exception of a few cases of bronchitis, we are at present very healthy." No exciting cause can be assigned for the appearance of this isolated case but a distressing and engrossing mental impression which the patient had endured by the intelligence of the death of his sister by *yellow fever* in the Colonial Hospital, a short time previous to his attack. Can the intense action of the faculty of Attention, long sustained, generate the peculiar morbid processes of yellow fever? or was the poison already wafted into the interior, and present, but too feeble to take effect, till the energies of the mind co-operated? A case not unlike this occurred in Berbice. The following is my note respecting it:—

"*Yellow Fever induced during the Epidemic Period, probably by the Action of the Faculty of Attention.*—Last week, Mr. S——n, who had charge of Mr. S——y's drug establishment in New Amsterdam, Berbice, died of yellow fever on the seventh day of his illness. Delirium and black vomit before death. He was a young Englishman, a *resident of the colony for five years and a half.* On the 12th of February last, he wrote a letter to Mr. S., in which he deplores the ravages which he understands were committed by the epidemic outbreak in Georgetown, and hopes that the disease will not extend to Berbice, as Dr. C. has informed him that he is just the very subject for yellow fever. Mr. S. wrote back an assuring and comforting reply; but which was never acknowledged. About six weeks ago, when cases appeared in Berbice, he again wrote to Mr. S. in the same desponding mood and manner. During the prevalence of the disease there he was even obtrusive in his visits to the hospital and dead-house, and dwelt with solicitous discrimination on the various post-mortem appearances, such as the different shades of colour in the liver, &c. It seemed as if the idea of yellow fever had taken complete possession of him as a fascination, and elaborated the fatal phenomena of the disease.—24th August, 1852."

May the early effect of exposure, in the cases of George Philips and William Smith, before referred to, be not due in part to the spectacles of disease which of necessity they witnessed in the wards, and the mental impression consequent thereon? Among the exciting causes of yellow fever may be mentioned the presence in the body of other febrile and irritating affections. A paroxysm of intermittent fever would sometimes set the morbid train in motion. I have notes of one case in which '*Rose*' (our colonial term for *lymphatic* inflammation), which is usually attended by one violent fever paroxysm, induced a mild attack in a native mulatto. The primary and secondary fever of small-pox also seemed to excite it. In illustration of this, I copy the following notes from my memorandum-book:—

"On the 6th inst. I was called by Mr. M'F. (engineer), who resides in a low, unwholesome part of Lacy-town (George-town), to see his little daughter, about four years old, white (creole), who had fever of two or three days' duration, but not severe, for which the father had purchased and given her 'Stable's Worm Mixture,' an American nostrum, of a pellucid syrupy appearance, the day previously, supposing her ailment to have been occasioned by worms. A dead lumbricoide (an entozoon present perhaps in every child in the colony) was passed. However, her fever continued, and when I was called in her tongue was red tipped, but there was no suffusion of face nor injection of eyes, and the fever very much abated after she vomited the contents of the basin which was at my visit shown me. This basin contained about three ounces of apparently genuine black vomit.

I prescribed bicarbonate of soda and creosote, and when I returned in the evening I found that she had vomited only once, and that consisted of several ounces of clear acid fluid (white vomit), with a mere sprinkle of snuff-like black vomit. Next morning all the gastric and fever symptoms were quite gone, but her face and body were moderately covered with small-pox (a disease which is very prevalent in town, particularly Lacy-town). The residence of Mr. M'F. is in the immediate neighbourhood of that of Mr. Charpentier, whose children and niece from the Upper Essequibo suffered so severely. I regret I did not think of examining the child's urine for albumen on the day I first saw the case. She is now running about with a mild small-pox eruption, and to-day I requested the mother to make the necessary examination, and explained how.—9th of November, 1852."

"*Another Case of Black Vomit excited by the Fever of Small-Pox.*—Mr. M'F.'s eldest son, aged ten, was taken with fever the day before yesterday. There are two cases of small-pox in the second house to windward of Mr. M'F., and his youngest child is passing through the disease in a mild form. Yesterday the boy had epistaxis; last night, bilious alkaline vomiting, but not a red tongue. To-day clear acid vomiting, with specks of incipient black vomit, and tongue fiery. Urine is not coagulable. To-morrow I expect the small-pox to appear. None of the family have been vaccinated.—13th of November."

"The second son, aged nine, has fever this evening, for which I have given him the resolvent dose in proportion to his age.—13th of November."

"Yesterday evening, after my visit, the eldest son again vomited the clear acid fluid, with specks and streaks of black vomit. Some efflorescence of face, and fleabite-like spots on legs and back, to-day. Still some fever. The younger son, to whom the resolvent dose and oil were given yesterday, is quite free of fever this morning. The eldest daughter, now the last of the family, twelve years old, got fever this morning, and I have prescribed for her the resolvent dose. The eldest boy's urine is still non-coagulable. There has been no yellow suffusion.—14th of November."

"The eldest son's small-pox is developed to-day. The eldest daughter's fever has been extinguished by the dose.—15th of November."

"*The Resolvent Dose aborting both Yellow Fever and Small-Pox.*—There has been no return of fever, nor a trace of any exanthem, in the two cases of M'F.'s children to whom the calomel and quinine were given. They are running about in perfect health. The two cases in which this treatment was not adopted are going on to maturity, but mild. This is an extraordinary *dénouement*. There can be no reasonable doubt of all their fevers having been identical, and originating from the same compound causes.—17th of November."

"To-day I was again called by Mr. M'F. The boy whose fever seemed evidently aborted, got it again yesterday. There has been no separation of the family, and the two cases of small-pox have been allowed to run their course, the whole family sleeping in two small adjacent rooms, the one having constant communication with the other, and the children all together in the same room. I again prescribed the resolvent dose, but as the fever has been allowed to run on full twenty-four hours, I can scarcely hope to have it aborted again. When I went to see him I found him lying in the same bed with his brother, who has now got the secondary fever. Another cause of my visit was to see Mrs. M'F., who was delivered this morning of a dead child apparently full-sized, but which she reckoned only at eight months. Mrs. M'F. had had small-pox about twelve years ago, and was complaining much of ill-health (*malaise*) about ten days ago. She thinks she did not feel the motions of the child for the last week. The cuticle was separated off the entire body of the infant, except the hands. But there was no pustular eruption that I could notice. Was it the small-pox poison, circulating latently in the mother, which destroyed the fœtus in utero?—28th of November."

"The small-pox exanthem has showed itself to-day, chiefly on the forehead, and a few on the hands. The fever is quite gone. The resolvent dose was thus too

late. There has been no black vomit, nor irritability of stomach.—28th of November."

"A few (four) vesicles, with central depression, have shown themselves to-day on the face of Miss M'F. They were not preceded by fever, but are evidently variolous. In the case of this family I think the power of the resolvent dose is distinctly seen.—3rd of December."

Cases of pneumonia, and even bronchitis also, have appeared to rouse the latent poison of yellow fever. This opinion of the effects of febrile and irritant diseases developing the epidemic influence is derived from numerous observations. In two cases attacks were induced from the irritation of passing a bougie. In one case in the Seaman's Hospital the pain and irritation of a whitlow appeared to set the morbid process a-going. The case of the head cook, with orchitis, has been already noticed. In the case of Philips, before referred to in this chapter, may not the analogous pathological condition of the eye, or the spirits of turpentine which he had been using internally, have contributed to the early evolution of the epidemic symptoms? It is possible that the *sea sickness* from which Chandler suffered may have had a similar effect, in the development of the Barbadoes malaria, against which he had heretofore been proof. In one case a relapse was evidently induced by the local and constitutional effects of *alcohol*. The particulars of the case are as follows:—A young man, named Cherry, of the *Maria*, aged seventeen, was attacked by the epidemic, but being the son of a ship-master in the same employ with the captain of the *Maria*, he was sent to private lodgings to be treated on the 8th of September, 1852. The fever and frontal headache, which had been very severe, were completely removed by two doses of the calomel and quinine. The eye, however, remained slightly injected, and a few fungiform papillæ continued very red towards the point of the tongue. I watched him for two days, and finding that notwithstanding the appearance of the tongue and eye, he seemed quite recovered, and had appetite, I left him sitting out on the gallery of the lodging-house. On the 11th, however, I was called back, and learnt that at midday he had been suddenly seized with severe supra-orbital headache and violent fever without rigors; I found him very flushed in the face, and stomach very irritable. He rejected instantly two successive doses of medicine. The turgescence of the face was so great that I opened the temporal artery. Convulsions came on about eighteen hours after, which lasted several minutes, and affected chiefly the right side, and a loss of consciousness remained for some time afterwards, and delirium supervened at night. At the same time, the sparse injected papillæ passed into an uniform crimson edge and tip. But the urine was not coagulable during the first thirty-six hours of this relapse. After forty-eight hours it was bloody and very coagulable, and he died on the morning of the 15th, having vomited and purged black vomit all night previously. He confessed on the day of relapse that he had drank a large quantity of "high wines," out of a bottle of it which was in his room for cooking purposes, mistaking it for rum, and that instantly he was taken ill. Yet total abstinence from alcoholic drinks during the epidemic seemed to yield no protection whatever. Perhaps the largest proportional mortality in the shipping during the exacerbation of June, 1852, was on board of the *John*

Bunyan, a temperance ship. All hands were sick, including the captain, and six of them died. The captain, after his recovery, was much comforted by the moderate use of alcoholic drinks, which he took under a medical dispensation. The *Emily*, also a temperance ship, suffered very severely at the same time; and the *Janet Wilson*, although in her the *mortality* was not unusually large.

The stench of *bilge water* seemed sometimes to be an exciting cause. The master of the *Chevalier* voluntcered the following information:—The pumps, he said, required overhauling, and one was brought on deck. The carpenter was told to put in his hand at the end of the pump, and haul out the filth which had obstructed it. He did so, and the stuff removed was very offensive. The carpenter complained and sickened immediately after, and died of his attack. He added, that the mate, then ill, also sickened after being engaged in work about the pumps.

One of the most favouring causes of the action of yellow fever poison was *infancy*. The constitution of the new-born or young white creole was highly susceptible. He or she was truly in the category of new-comers. Not only did the first fatal cases in town occur in children, but they followed numerously and repeatedly, as in the family of the Rev. Mr. C., and Mr. W., and others, too many to recapitulate. As these infants and children were not exposed to some of the physical and moral influences which favoured the attack on adults, their high susceptibility can be imputed to structural difference only.

The instance of Mr. Vervestein would lead to the supposition that the tendency to yellow fever may be *hereditary*. Many facts came to my knowledge which showed that *family predisposition* for this disease exists and is evinced under varieties of exposure. It was noticed in several cases that a scorbutic diathesis or sponginess of gums in the individual attacked, prognosticated the worst results. But the great predisposer—the pabulum on which the epidemic revelled—was the organization of the white who had recently arrived from an elevated or mountainous country beyond the tropics. Between the 1st of January, 1852, and the Christmas-day following, the total admissions to the colonial hospital were 3712 in-door patients, of which 662 suffered from yellow fever; while during the same time, in the Seaman's Hospital, there were admitted 1308, of which 1049 were cases of yellow fever. Thus in the former, which contains and represents the resident population, the ratio of admissions was only 18 per cent.; while in the latter, which represents the European and North American transient population, the ratio of yellow fever admissions is upwards of 80 per cent. And even this disparity would be greatly increased if the Portuguese immigrants who have but lately arrived in the colony, be struck out of the computation, for of this class does the great majority of admissions for yellow fever to the colonial hospital consist.

On the other hand, in looking for the causes which operate in retarding, or mitigating, or entirely shielding the action of the yellow fever poison, in the infected localities, we find that cheerfulness of mind, active but not laborious occupation, regularity of habits, and avoidance of night air, sustain the tone of health, and militate against the inroads of the prevailing disease. The appearance of the eruption of small-pox seems to supersede the yellow fever poison. The presence in the system of evacuant

diseases, such as the advanced stages of phthisis when the tubercles have softened, and even gonorrhœa, seems to have a retarding power. Several instances in the hospital were observed of attacks supervening on the *healing up* of the discharging surfaces of burns, scalds, and wounds. On board the *Glenelg* and several other vessels, the free use of Sir W. Burnet's disinfectant fluid was found inefficacious. On board the *Susan*, in March and April, 1853, the use of chlorine gas seemed to arrest the infection. Four of her men sickened in succession and were taken ashore, and though they received the utmost attention, all four died. After the fourth case, the ship was well and repeatedly fumigated till the time of her sailing, three weeks afterwards, with chlorine gas. No other case occurred on board. I am not aware if the experiment was ever repeated, but it deserves to be.

Of all the protections, that of *complexion* was paramount. When the ships' crews were disabled by sickness (and that was in the majority of instances), their places were supplied by negro sailors and labourers. On board of many vessels, black labour alone was to be seen employed, yet among these labourers and stevadors a case of yellow fever was never seen. If to the table of thirteen months' admissions to the hospital, already given, be added a classified census of the population of the colony, information is furnished which enables us to arrive at something like precise knowledge on this subject. The following is the additional table:

Population of British Guiana on the Night of the 31*st of March,* 1852.

Natives of British Guiana	86,451		
Natives of Barbadoes	4,925		
Natives of other West India islands	4,353		
African immigrants	7,168	+ 722 = 7,890	Added by immigration till 31 Dec. 1852.
Madeirians	7,928	+ 2,363 = 10,291	
Coolies from Calcutta and Madras	7,682	+ 3,296 = 10,978	
Old Africans	7,083		
English, Irish, Scotch, Dutch, and North Americans	2,088		
Not stated	17		
Aborigines, estimated at	7,000		
Ship's company of H. M. S. *Inflexible**	150		
Merchant seamen	295		
Strength of 2nd West India regt.	369		
Strength of 3rd West India regt.	298		
Strength of 72nd Highlanders	187		
Total on 31st of March, 1851.	135,994		
Additions by immigration till 31 Dec. 1852	6,381		
	142,375	less 337 sailed = 142,038 on 31 Dec. 1852.	

* The white troops were removed on the breaking out of the epidemic. The steamer *Inflexible* sailed before that epoch. The European and North American population are white. About an equal number of the native population, or creoles, may be estimated in the same category. The Madeirians and merchant seamen (although sometimes the cook or steward on board the merchant ships are negroes) may also be included in it. Deducting then the white troops and crew of the *Inflexible* from the grand total, it will appear probable that *on any day* in the year 1852, the relative proportion of the *white* to the *dark* races was as 14,726 to 127,276; while the admissions to the public hospitals for yellow fever were 1947 of the former to 59 of the latter.

From this it would appear that the liability of the white races to yellow fever, as compared with the dark, is as 13·19 to ·00004. But this would be rather an over-estimate of the risks of the whites, for although the calculation is correct for one day, it is not for the whole thirteen months. During the year 1852, 7670 seamen, the crews of vessels, arrived at the port of Georgetown. If we add one-twelfth to this sum, it will make a total of 8309 persons, estimated all as white, who for a longer or shorter period were exposed to the epidemic influence. This number should be added to that of the white population exposed, and the per-centage of liability will be as follows—*whites*, 8·430; *darks*, ·00004. This computation is irrespective of the effects of *residence* on the constitution. But the numbers afforded by the census returns are sufficiently great and detailed to authorize a purer and more ultimate analysis of the effects of complexion, or in other words, *cutaneous organization* on the liability to yellow fever among the population of the colony. We find that of 7890 African (black) immigrants, *none* contracted yellow fever.

Of 9278 West India Islanders (black and mulatto), 15, or ·16, contracted yellow fever; of 10,978 Madras and Calcutta Coolies (black, but fine-haired), 42, or ·38, contracted yellow fever; and of 10,291 Portuguese immigrants (white), 698, or 6·2 per cent., contracted yellow fever. The *aborigines* all reside in the interior, and out of the infected localities.

From the foregoing the importance of the skin, or of that constitution of the body which is associated with varieties of the dermal covering in the etiology of yellow fever, is at once apparent.

CHAPTER XV.

During the non-epidemic period, the reports of fever cases in the hospital books were very brief and simple. The name of the patient, his age, native country, and a few other statistical facts, and the type of his fever, were entered. Afterwards followed the prescription of the anodyne draught or aperient mixture, and six grains of quinine, in solution, every hour till six doses be taken, during the intermission. The next and last entry was, in ninety-nine cases out of a hundred, as follows,—"Cinchonism, apyrexia, purged, discharged."

When the epidemic again broke out, the old formulas of reports and prescriptions became obsolete. In private practice a similar revolution occurred. Indeed, during the epidemic period, the anodyne draught (spir. æther. nitros. ʒj., sol. acet. morph. gtt. xv., aquæ ʒij.), which, in simple uncontaminated intermittent fever, acts on the fever and fever aches like magic, became positively dangerous, and simple quinine solution was not always successful in preventing the recurrence of paroxysms. The staple prescriptions of physicians were thus entirely altered since the advent of yellow fever.

When the epidemic poison was in moderate intensity or quantity, the results of treatment were highly gratifying. At such times when the disease was recognised and treated early, the chances of aborting the seizure were very favourable and decisive. If, in addition to this medium intensity of the epidemic influence, the favourable conditions of residence for a considerable period, or a cross in the blood, were added, the prescription was given with confidence of success. But at times, when the

system seemed thoroughly saturated with the poison; when every mucous tissue was more or less irritated by it; when no auxiliary or exciting cause was required; when the attack was violent on many points, and spontaneous; when, in fact, the exacerbations of the epidemic became pestilential, medication was powerless, and the morbid processes terminating in death were scarcely, if at all, modified or interrupted. The prime object of treatment, however, was to *abort the attack*. If that failed, after one, two, or three doses, although still much could be done in putting the patient in the best condition for sustaining the struggle and keeping off intruding complications, there was little room for active interference on the part of the medical attendant. Early attention to first symptoms among the susceptible was of priceless value in saving human life. Numerous instances of this occur in my notes. The following comparative case will illustrate the point:

"The ship *G.*, and the barque *M.*, both of Bristol, lay within cable length of each other: both within the infected locality of the season. The latter has been about a week longer in the harbour. The master of this vessel is very attentive to his men, and quietly and without exciting alarm ascertains the state of their health twice or thrice in every twenty-four hours. The master of the former is seldom on board his vessel. The master of the *M.* has informed himself of the premonitory symptoms and the treatment to be at once adopted on the instance of their occurrence, especially when they supervene at night, and until professional aid can be procured. The consequence is, that of ten cases which I have seen on board his vessel, all have been aborted; while in the other vessel three deaths have taken place out of four cases, and the fourth case, which is recovering, was brought to me a few hours after the attack, the master having then been aroused to the necessity of early measures."

"C. Bush, of the *Superior*, was admitted to the Seaman's Hospital on the 5th of April, 1853. He had accompanied a messmate the day before, who was admitted with marked symptoms of the prevalent disease, but which was soon aborted. Bush himself complained of frontal headache then, and I recommended him to remain and rest himself in the hospital for twenty-four hours (that he might be further observed). But as he then had no fever, and there was no capillary irritation visible on ordinary inspection; and as he had a small boil between his eyebrows, which might cause a pseudo headache, he was allowed, at his own request, to return to the ship, with a poultice to his boil. Next evening (on the 5th) he was brought back with all the symptoms of an attack violently developed, and died, having had black vomit on the second day after admission. Thus was twenty-four hours of valuable time lost, and thereby probably a human life."

The compound which is represented in the annexed cases by the symbol of 20 + 24, and which constitutes the aborting or resolvent dose, consists of twenty grains of calomel added to twenty-four grains of quinine. The *mistur. magnes.* there referred to, and which was so frequently used as a substitute for castor oil, and following the first dose of the resolvent, is composed of two drachms of carbonate of magnesia to two ounces of sulphate of magnesia, in eight ounces of peppermint water. As quinine in some persons induces nettlerash (from, I have reason to believe, the mechanical irritation of its spiculæ), it should, when exhibited in the solid form, either alone or in combination, be finely triturated before it is mixed with the calomel and administered. This fine division will facilitate its solution, and prevent its getting involved among the rugæ

and plicæ of the mucous membrane of the stomach. As a vehicle, I believe that syrup, or honey, or pap, are equally good, and superior to any other vehicle. Owing to the bitterness of the dose it was administered at one time in *capsules*, of which four contained the dose. But this mode is open to the obvious objection, that the physician and patient are at the mercy of the manufacturer, who, if dishonest, may include some cheap, or effete, or delcterious compound, as a substitute for the medicine intended. Another grave objection to the use of capsules was their slow solution, and they were finally disused at the hospital. If the stomach have a strong repugnance to the dose when mixed in syrup or pap, it then may be swallowed wrapped up in *wafer paper*. But I preferred that the dose actually touch and pass over the mouth and œsophageal surfaces, on its passage to the stomach. In preparing the irritable stomach for the reception of the dose, *creosote* had often an admirable effect; many instances of which will appear in the appended cases. The aborting doses were repeated at intervals of four or six hours; but at the time when the second dose was due, the purgative (either oil, or two ounces of magnesia mixture) interrupted the succession. Of the number of doses which have been administered in any individual case, I believe four has been the limit. In the case of Manning (Seaman's Hospital, 1st of Oct., 1852) that number had to be given before the attack was aborted, although in convalescence only a mere haze of albumen appeared in his urine. Sometimes, but rarely, the dose induces early and hyper-cinchonism; and, on the other hand, such a tolerance of it sometimes exists that, as in the case of Nichole (Seaman's Hospital, 28th of Jan., 1853), four doses did not induce cinchonism. When the disease is taken early, or the epidemic pulsation is moderate, one dose followed by the purgative will generally be adequate to its removal. In practice, sometimes, the intermediate purgative is beneficially omitted, and the *coup sur coup* system answers. Thus, on the 9th of December, 1852, in the afternoon, I went on board the *James Erving* to see the mate, who was suffering from yellow fever from the previous night. He had taken a purge in the morning, and as I could have no opportunity of seeing him again that evening, I left a prescription for two doses—one to be given at once, and the other eight hours afterwards. Going back next morning, I found he had retained both doses, and the symptoms were each and all dispersed. The powder had acted freely on the bowels. I did not require to see him again. We found the aborting dose less efficient against relapses than primary attacks; hence the urgent need for avoiding the infected localities for a considerable period after recovery—a condition, however, impossible in the case of the unfortunate seamen.

One of the earliest and most uniform effects of the dose in the treatment of aborted cases, is the removal of the headache symptom. It is likely that this symptom properly belongs only to the early stages of yellow fever, and that its tendency is to subside spontaneously; but its departure is unquestionably hurried by the agency of the medicine, and the first or second dose is generally adequate to its removal. While the same amount of the compound given in small and frequently repeated doses would infallibly cause salivation, such an effect is of the rarest occurrence in the large doses, and when it has happened, never, that I have seen, but

mildly. I have prescribed it, without injury, to females far advanced in pregnancy; and to my own infant, three and a half months old, in a similar dose, proportioned to the age, and found it attended with no practical inconvenience of any consequence. The *modus operandi* of the dose in aborting yellow fever, probably, is not by the constitutional effects of mercurialization. Calwell (Seaman's Hospital, 25th of March, 1853) while accidentally salivated for another malady, got a violent attack, *which was aborted by the usual method.* Three doses were in this case required and found sufficient, and without any increase of the salivation.

The aborting dose should be used as early as possible. When a state of apyrexia is induced, it may be relinquished; the end is attained: but if the urine has become coagulable, or the epithelium of the tongue has begun to peel, it is of no use pushing it further, the time for its administration is past, and subsequent to this it will be a noxious irritant.

From information which we received through the surgeons of the West India Mail Steamers, we could see that the use of calomel and quinine in the treatment of the epidemic was not understood, or rather was completely misunderstood, among the West India Islands. We were told that it was pushed on in various doses and proportions, through all stages, and whether the stomach retained it or not. Nothing could be more injudicious. Its benefits are confined to the first and early stage; and though, if the case run on, some mitigating effects may flow from its previous use, still it is for *aborting the attack completely and at once* that it is prescribed and is suitable. Sometimes the disease is incompletely aborted,—that is, although the disease does not proceed to the second stage, a certain amount of febrile action still continues after the resolvent has been pushed to a reasonable extent. It was the practice then to give half an ounce of camphor water and spirit of Mindererus every three or four hours, till the skin became cool and soft. Should, however, the stage of acid elimination supervene, this medicine is stopped, and small doses of bicarbonate of soda and nitrate of potash substituted. The *rationale* of this treatment is not so obvious as it would appear. The acid elimination seems in many cases a salutary act, the disease sometimes terminating with that stage; and the fever very often and so suddenly ceases, as to impress the belief that the phenomena are associated as cause and effect. Then why use antacids? Perhaps until more is known of the part which the acid elimination plays in the pathology of the disease, the medical practitioner should use his test paper frequently, to enable him to know when an excess of alkali has been used, that the quantity of soda, potash, or chalk may be kept up to the point of neutrality only. For if it be a critical evacuation, but salutary only when moderate in quantity, an excess of the medicine should be avoided, as it is well known in chemistry that the presence of a free alkali is apt to induce an opposite condition where there exist the elements to bring about such combinations. It is therefore to some extent in the power of the practitioner to command this symptom. We used the compound of nitrate of potash and soda in the proportions of from five to ten grains each for a dose, because we believed we saw it improve the condition and comfort of the patient, and speculated that among its other effects, the benefit might arise from its gentle action on the kidneys, and the relief of that uneasiness of the

stomach which the presence of free acid in its secretions always creates. The selection of the alkali is not indifferent. Except the nitrate in small doses, potash and its salts were found objectionable. Liquor potassæ and the carbonate, unless excessively diluted, possess a causticity which render them difficult of tolerance, and sometimes distressing to the denuded mucous membranes. Magnesia and chalk are sometimes eligible; but the most generally suitable was the bicarbonate of soda. When the mucous surfaces, as indicated by the tongue, were denuded of epithelium, the use of *gum water* was decidedly beneficial. It lubricated, defended, and soothed the raw surfaces. The strength was generally three drachms of the purest powdered gum arabic dissolved in six ounces of cold water, and a table-spoonful of this given every one or two hours. The patient at last gets tired of it; but for thirty-six or forty-eight hours of the most critical period of the disease, it is used without dissatisfaction, and then can be substituted by, or alternated with, arrow-root pap. When the heat of surface was ardent, the *wet sheet* or *blanket* was used for the reduction of temperature by evaporation, with frequently very good effect. But in the late stages of the disease, when the skin was cool or cold, the patient seemed to have an instinctive craving for its reapplication, and frequently asked to be put into it. There would appear to be two causes for this feeling. We find it to exist in cases in which black vomit has been copious, and the associated thirst distressing. Also, as in the case of Tomlinson, where there has been no black vomit of any consequence, and the breath is highly ammoniacal. In the former case the stomach ceases to be an *absorbing* viscus in anything like the proportion of its secretions and transudations. The skin is therefore employed in reducing the crasis of the blood by the absorption of water, as ship-wrecked mariners are said to quench their thirst. But not only does the skin afford an inlet for the imbibition of diluting fluids, but the softening of the cuticle would seem to afford an additional outlet for the noxious elements of the circulation; and it is probably in this direction we must in future look for auxiliary means of relieving the blood of its poisonous, metamorphosed, and effete constituents, the onus of which is now thrown on such vital organs as the stomach and lungs. At one time, the heat of the surface was so ardent and persistent, that the wet sheet failed to reduce it effectually. For these cases, I once or twice only tried the effects of tobacco injection.

The *food* during the course of yellow fever should be of the blandest description: chicken tea, arrow-root, sago, and barley water constituting the chief articles; and these should be taken when the stomach is at all irritable, in minute quantities at a time. This rule also applies to drinks of all kinds. The patient is greedy for a large draught of fluids; but by sucking them through a glass tube of small bore, or by the tea or table-spoonful, they are much more likely to be retained. A cold infusion of oatmeal was found an agreeable drink for the Scotch seamen, of which they did not seem to tire. A dislike of sweets was observed among the patients, and when lemonade was asked for, the usual quantity of sugar was objected to, probably from its rendering the liquid too dense for ready absorption by the stomach, and therefore less quenching. *Tea* was found so uniformly to disagree with the patients, and cause vomiting, particularly in the advanced stages, that at length it had to be expunged from the yellow

fever dietary. Dilute alcoholic drinks were given freely, and with good effect. Unfortunately, the quality of the hock wine to be obtained was much inferior to that used in the former epidemic; and from its acidity, frequently disagreed with the stomach, and fell into disuse. Where brandy could be obtained pure (tolerably free from acidity and fusel oil), and was well diluted with water, that spirit answered every indication. Sometimes the effervescing wines were relished and retained, but they are very liable to the objections of containing foreign matters, and the products of mismanaged fermentation.

During the course of the disease, *auxiliary treatment* was required to meet contingent symptoms. This was embraced chiefly in the use of local and general blood-letting, croton oil, morphine, ether, vesicatories, hydrocyanic acid, and the creosote before referred to. Cupping, leeching, and blistering were found useful in relieving the primary head symptoms and irritability of stomach, when applied respectively to the nape of neck or epigastrium. Tenderness over the liver seemed also benefited by these applications; but I cannot say I have ever seen any benefit resulting from their application over the kidneys, with the view of relieving that congestion of which albuminosity of the urine and suppression are the indices. In only one instance have I seen strangury follow the application of blisters in this malady, and in that case it seemed to exercise no injurious effect. Seeing that herpes labialis was a favourable indication, and arguing that their vesications might be beneficial from their situation at the termination of the mucous surfaces, we created on several occasions an artificial herpes, by brushing the lips and parts around the mouth with the acetic ether-infusion of cantharides. This operation, however, was without results. When the primary reaction was violent, and the face was turgid, and the head symptoms severe, arteriotomy was performed, and with benefit. In a few such cases, and when the patient was young, strong, and full-blooded, and where the dynamic congestions were so violent that the vessels yielded to the turgescence and impulse, and blood-corpuscles without tube-casts, or even but a haze of albumen, was present in the urine, the arm was opened, and free bleeding relieved the tension of the vascular system. In such cases, convalescence was slow and unsatisfactory, but the immediate results had been beneficial. In general, the bowels responded easily to the action of mild purgatives; but a cluster of cases occurred about fifteen months after the commencement of the epidemic, in which *croton* oil was required to follow the resolvent dose. Hydrocyanic acid was supposed beneficial in a few cases in abating the primary irritability of the stomach; and being easily taken, may be borne in mind by the practitioner, as a variety of such resources are at times required. *Ether* was frequently attended with marked advantage in removing or abating the distressing symptom, hiccup; but we used it also as a diffusible stimulant, and where acceptable to the patient, is fully equal to brandy for that purposes.

Of all the auxiliaries which must be occasionally impressed into the service of the patient, by far the most important is *morphine*. I am inclined to think that the type of the present epidemic tolerates that drug more easily than the last; but there is no doubt that its management is better understood now than then. Its administration, however, still involves more knowledge, discernment, and judgment on the part of the

practitioner than any other drug he has to deal with. In the present epidemic, the most salutary effects were observed from its use in the beginning; but a number of cases occurred in which it was so manifestly detrimental, that its use was about being relinquished again. In some of these cases in which it was injurious, its first effects for some hours seemed favourable; and for a considerable time, no criterion was known for its administration. Various conjectures arose, at the same time, as to the mode of injurious action both of morphinism and hypercinchonism. Following the cue of Frerich's theory, it was supposed that the drugs supplied some element to some other casual element in the blood, as *emulsine* and *synaptase* converts harmless amygdaline into poisonous hydrocyanic acid, or a diastase quickens starch. As quinine and morphine are alkaloids, and contain nitrogen, and are very complicated in their constitution, and possess high combining powers, the hypothesis was for a moment feasible. But I suspect that the injury frequently arising from the use of morphine is chiefly due to its action on the secretions of the kidneys. It impairs that function; and where the march of symptoms is already verging on that of urinary suppression, although the tranquillizing effects of the drug may be pleasant for the time and well marked, it indirectly induces head symptoms, and adds to the uræmic poisoning. The rule therefore would be, *not to give it when there is suppression or tendency to suppression.* Of course, if the restlessness or sleeplessness or suffering is extreme, it becomes a question for deliberation, whether, even in suppression or tendency to it, the relief which is sure immediately to follow the dose of morphine will compensate for the jeopardy of life. The necessity must be extreme indeed that would justify, for present ease, the surrender of the smallest chance in favour of ultimate recovery. Its beneficial effects are most visible and unqualified in those cases wherein the disease has been imperfectly aborted, and which, after a few doses of the aq. acet. ammon. and camphor water, will induce a good night's rest, out of which the patient awakes free from disease. Morphine is perfectly safe while the urine is non-albuminous. The effect of yellow fever on the system is to make it sensitive to narcotics. Cases of delirium tremens with a taint of the epidemic will not bear that liberal use of opiates of which it is normally so tolerant; and a dose such as that which the anodyne draught contains, is too much for yellow fever, though never found so for intermittents. After many observations, I have come to the conclusion that, for an adult, eight drops of the solution of the acetate (one-fourth of a grain) should be the maximum dose, and should rarely be repeated within twenty-four hours.

The "smouldering form" of yellow fever is best treated by rest, the recumbent position, cool drinks, and abstinence from any but the lightest food. The patient, however, should be closely watched, although interference is seldom required, the curative and conservative power of nature being adequate to the perfect restoration of health in almost all these cases. Inflammatory complications were treated on general principles; and in pneumonia, the tartrate of antimony was borne well.

Before concluding, it may be instructive to notice the results of a few unsuccessful experiments, undertaken during the course of the epidemic. The chief of these was the trial of the use of belladonna as a prophy-

lactic. Remarking the close analogy of yellow fever with scarlatina, I drew up a representation to the Board of Health, and suggested an universal distribution of the drug among the seamen from the moment of their arrival in harbour, in the same doses as had been employed in Europe for the prevention of scarlatina. It was styled "the protection fluid," notices of which occur in the annexed cases. The Board, with the utmost alacrity and zeal, took up the subject, and carried out the experiment for almost two months as efficiently as was possible to be done. Among the patients who presented themselves afterwards at the hospital, I never observed on the skin or eye any of the specific effects of belladonna; but there is no doubt, from the exertions of the Board, and the spirit of the ship-masters and mates, it had been, with very few exceptions, regularly, steadily, and for a prolonged period, administered. This prophylactic for about a fortnight obtained that spurious popularity for success which is not uncommon, and is the result of accidental circumstances. The intensity of the epidemic had suffered one of those periodical fluctuations before noticed, and the *post hoc* was mistaken for the *propter hoc*. But the mistake did not last long, and it was soon evident that the epidemic influence was wholly unaffected by the medication with belladonna.

Early in the epidemic I obtained from Dr. Thier some carefully prepared bisulphate of lime, and used it for black vomit, without any advantage resulting. A skilful chemist, Dr. T., under the impression, from certain observations made on himself, that an important link in the chain of morbid phenomena was an incapability in the stomach in oxygenating nutriment—in other words, of performing digestion,—suggested the use of pepsine in some of our cases. It was prepared and tried in five cases; all died. Of one of these only was a post mortem examination made (Gambling, Seaman's Hospital, 9th of April, 1852); and, although he had not had much vomiting before death, his stomach was eroded in great deep longitudinal stripes, and the lesions were more severe than I had ever before seen. It is clear that pepsine is injurious in yellow fever, and from the knowledge of this fact perhaps some light may fall on the obscure pathology of the stage of acid elimination.

In the beginning of 1853, her Majesty's vice-consul at Bolivar, Venezuela, becoming afflicted with the *cacoethes scribendi*, inundated the several West India governments and colonial newspapers with accounts of the discovery made by a Madame Orfila of a certain, sovereign, and infallible cure for black vomit, effected by a plant indiscriminately named *verbena* and *vervena*, which is very abundant. And of this plant a specimen *leaf* was always forwarded, which, like the brick, displayed as a specimen of a house, in the old joke of Theophrastus, was intended to convey full and complete information of the plant. There were, however, often *two* leaves sent, one larger than the other. The one was stated to belong to the *male*, and the other to the *female*, verbena. The name of Orfila, although in this case it designated an ignorant old half-blood Indian woman, no doubt promoted the renown of this new remedy. One of the worthy consul's circulars, of course, reached Guiana, and his excellency the governor, as it came in official garb into his hands, very properly referred it to the surgeon-general, with instruction for a trial of

the remedy, and a report of results to be forwarded to the Government secretary's office. The statements of the circulars contained abundant evidence of scientific ignorance, and were replete with absurdities. But still it was possible that a savage or an ignorant person might stumble on a great medical discovery—the cinchona, for example—and be unlucky in the expounder. I therefore set about obtaining information which would lead to a knowledge of the plant really meant. As the genus verbena is pretty extensive, including even East India teak in its family, it was not so very easy to determine the species and variety so highly recommended. At length, having obtained an entire plant of what was admitted by those, who should know, to afford the genuine remedy, I discovered, through Mr. W. H. Campbell, whose name is a sufficient guarantee for its accuracy, that this treasure was the *stachytarpha Jamaicensis*. The nauseous and disgusting compound was prepared and administered precisely according to directions, and, it need scarcely be added, unsuccessfully. Its want of success, however, was less matter of concern, as about this time the worthy vice-consul, ever anxious, as he declared himself, for the welfare of mankind, announced through the newspapers the discovery of another sovereign and infallible remedy for the same complaint.

It requires apology for referring to this ridiculous affair, but the verbena of Madame Orfila has been, I perceive, the subject of grave conversation in the London Epidemiological Society.

CHAPTER XVI.

There is a material link in the chain of evidence yet to be supplied before the following definition can be dignified by the epithet of theory. It must be demonstrated to be a fact, by submitting the arterial tubes and capillaries to microscopic examination, that the epithelial covering of these vessels does really undergo the desquamatory process which is so noticeable in the open mucous tissues. This has not yet been attempted, and till accomplished the generalization now offered, though it explains the chief morbid phenomena and their order, can be received only as an hypothesis. *The efficient cause of the disease known as yellow fever is an aërial poison, probably organic, which requires a certain temperature for its generation and existence, and affects special localities and persons. This poison attaches itself to the mucous surfaces of the human body. One of the primary effects of such contact, when the quantity is adequate, is to rouse the system into febrile reaction, and to excite through the stomach and intestines an effort to expel the noxious agent. There is reason to believe that this compulsory effort is sometimes successful unassisted, but is materially aided by the action of certain medicinal substances. In the event of the expulsory effort being unsuccessful, the effect of this poison is to act destructively on the epithelial structures of the body by inducing a specific irritation in the basement membrane, by which, and by allied consecutive lesions, the arterial and capillary tissues are impaired, the viscera become congested, the blood thereby contaminated by suppressed secretions, and fatal hæmorrhages ensue.*

THE END.

APPENDIX.

THE link in the chain of evidence referred to by the author as deficient when he wrote the concluding section of his paper, appears to me to be supplied by his later researches—those of which he gives an account in the following letter:

George Town, Demerara, March 8th, 1856.

MY DEAR SIR,—I beg to enclose for your examination a small fragment of material which was expectorated by a seaman, Thomas Bailly, suffering from yellow fever in the Seaman's Hospital, on the 29th ult. The expectoration at the time of observation was of considerable quantity, amounting probably to an ounce. Some of it had a clear glairy appearance, and some was of rather an opaque white, and of a tenacious consistence. Mixed with this expectoration were several red spots, apparently minute blood-clots. On microscopic examination, the pale portion was found to consist chiefly of epithelium, but no cilia were observed on the cells, which were in general very perfect. Several fragments of broken capillary vessels were found mixed with it. When the red spots were subjected to examination, they were found to consist of bundles of capillary fragments, tinted of a bright pink or crimson, and without blood-corpuscles being present. Under the one-fourth and one-eighth inch object-glass of Ross, several of these capillaries were found to be colourless. I enclose a small portion of this material in tinfoil; and, lest decomposition should injure the specimen before it arrives at its destination, I have mounted a minute portion in Canada balsam, which is also sent. Although it is only a week put up in the balsam, I find that it has lost much of its brightness of colour already. The fimbriated ends are also injured. It would likely have done better in a glass cell preserved in Goadby's solution, but I feared that the thin glass of the cell would have been fractured in passing through the post-office. I hope, however, that between the two samples sent, sufficient may reach you to enable you to form a correct idea of its structure. On the 4th of last month, in the case of a seaman named Morrison (fatal), I for the first time observed the undoubted presence of broken capillary vessels in the excretions of yellow fever. In his case, also, it was first noticed in the expectoration. On all former occasions, epistaxis or bloody expectoration was looked on carelessly, as merely a manifestation of the hæmorrhagic tendency, and nothing was expected to be seen but blood-corpuscles under the microscope. These symptoms were therefore almost unheeded hitherto. On this occasion, however, some turn of thought suggested more particular attention to the subject, and the examination of Morrison's bloody sputa led to important results. I have since found the existence of broken capillary vessels one of the commonest phenomena of the disease. They are to be found sometimes in great abundance in the urine, in the alvine evacuations, in the white vomit, in the flaky sediment of the black vomit, in the bloody exudations and hæmorrhages from the mouth, and even on the blistered surfaces. In the flakes of black vomit, it is sometimes necessary to dissolve off the albuminous matter by a drop or two of liquor potassæ before they come fully into view. I

had often seen them formerly in the urine and black vomit, and other fluids eliminated from the subjects of yellow fever; but as in most cases they are colourless and empty when so found, I was wont to set them down as extraneous bodies, and suspected them to be fibres derived from the linen sheets and towels of the establishment. With this preconceived idea, they were of course overlooked and unrecorded. On turning up some old mounted specimens of "caddy stool" of the epidemic of 1851, I find these vessels still existing in them. The fragments of capillaries are found generally in single cylinders; I have seen, however, a few branched and bifurcated. Their tendency to break off seems to be at the *bendings*. The fracture is occasionally clean, but generally the broken end is split into filaments. A separation of filaments seems to be the mode in which the fracture occurs; and in many fragments, the length of which will occupy three or four times the field of vision of a half-inch object-glass, several partial fractures may be observed in which the tube at such points is split all round longitudinally, and a perfect sub-division is about to occur. At such points on the outer angle, and at the open ends of the capillary fragments, the *débris* of blood corpuscles is to be seen, and these sometimes form a little dossil which is seen connected with the tube of the vessel by fibrillæ. In the urine, I have seen some of the capillary fragments enveloped in the tube-cast material, but encrusted evidently with flat instead of spheroidal epithelium. I cannot observe in the specimens which I have now sent, any epithelial lining within the capillaries; and yet their calibre, I think, is such as would lead us to expect its presence, were they not diseased. In some of the specimens which I have kept of the same expectoration, epithelial matter is visible alongside of the broken capillary vessels, as if it, as well as the blood, had escaped from their cavities. Finding that ecchymosis of the conjunctiva, epistaxis, and some other hæmorrhagic appearances, are common in yellow fever long before the blood has apparently lost any of its fibrine; and finding that even when black vomit is established and the tongue is smeared with blood, the corpuscles are normal in appearance, I cannot but look on the textural lesion of the capillaries as a primary effect of the yellow fever poison, and as the *cause* of the congestions, ecchymoses, oozings, and hæmorrhages, and all their consecutive mischief. The phenomena of the present minor epidemic also corroborate the view that the poison attaches itself to the mucous membranes in the first instance. Its early effects seem to be local. The system is thereby inoculated, and the poison spreads to all the analogous tissues of the body. A general impregnation of the *circulation* in the first instance would be scarcely compatible with the fact of the slow, steady march of the pathogenic influence through the various organs of the body. I enclose the case of Thomas Bailly, as reported in our hospital case book. It will be seen how his attack commenced like a "common cold"—began in the bronchi, and how it gradually extended to the conjunctiva, mucous membrane of the mouth and fauces, to the liver and kidneys, and its final resolution. I may mention, in "reporting progress," that I have detected the glandular cells of the liver to be a common and very large constituent of black vomit. Their shape and size and tint, and the presence of minute oil-globules beside the nucleus in the epithelium, leave no doubt in my mind as to their identity. In the flakes of the black-vomit sediment, also, there is not much difficulty, with the addition of liquor potassæ, in distinguishing the bile-flakes from the blood-flakes.

I remain, my dear Sir, ever faithfully yours,

D. Blair.

Dr John Davy, F.R.S., &c. &c.

Relative to the morbid specimens referred to in his letter which my friend has sent me, and which I have examined with some care, I may remark that their appearance under the microscope accords well with his description; and in consequence, that I entertain no doubt that they are really fragmentary portions of vessels. The only substances much resem-

bling them that I am aware of, are filaments of cotton and flax; and from these I find they differ in not being acted on and dissolved in the same rapid manner by strong sulphuric acid at the temperature of the atmosphere: moreover, their diameter is different, and their general aspect is different.

Dr. Blair has come to the important conclusion, that the lesion of the vessels precedes and is the cause of the hæmorrhagic phenomena—in brief, that yellow fever is not a primary blood disease, having found that the blood corpuscles are not sensibly altered at an advanced stage of the disease, even after the occurrence of 'black vomit'—a fact I can confirm by the observations which I made on the blood during the Barbadoes endemic of 1847-48.—(See foot-note in the author's work on 'Yellow Fever,' p. 37.)

J. D.

Lesketh How, Ambleside,
April 2nd, 1856.

THE END.

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